NEW

Eclectic Medical Practice

DESIGNED FOR

Students and Practitioners

BY

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VOLUME I.

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PREFACE.

THE author is not aware that an urgent demand has existed for the publication of this book. Indeed, it was not prepared with the impression that the profession was in urgent need of it, or that it would prove a remarkable innovation in the medical world. Medical men will come and go and the world will move on, after these pages have passed into forgetfulness; and time will chronicle a newer practice—doubtless a better one—when the years have marked the inevitable progress of events.

The students of the California Medical College, to whom the author has been something of an authority on practice for the past sixteen years, have frequently importuned him to publish a work which would embody the substance of his lectures, that a text-book for ready reference might be had. To satisfy this demand the present volume was begun several years ago. Other duties, however, have delayed the work, and the prospects of its completion at an early date were so poor that at the request of several members of the Class of '99 a first volume has been published, the second to follow within the coming two years.

Special pains have been taken in the preparation of the text to dwell on the description of disease, that there may be no need of frequent reference to allopathic works for points on etiology, pathology, symptoms, diagnosis and prognosis. At the same time, it has been the author's aim to render the book as complete as possible on that which has been the peculiar merit of Eclectic works on practice in the past, treatment.

Attempt has been made, throughout the work, to give every writer from whom points have been drawn his just due, especially when anything appropriated appeared to be original in its conception. As appropriate credit has thus been given, it would

only be superfluous to reiterate here what the text will plainly show.

Credence has been given the teachings of bacteriology, on the ground that it offers the only reasonable theory for the spread of infectious diseases that has ever yet been advanced. Though there are those who scout its doctrines, unfortunately for their skepticism they are unable to offer another as good an explanation of the spread of contagium. Without its teachings we would be as sadly at sea as before. Undoubtedly, many of the theories of bacteriologists are sound, though there may be much to learn; and there may be some things taught which will have to be unlearned at a later day. Until a better explanation of the etiology of infectious diseases has been offered, however, I shall follow its lead, to a moderate extent, at least.

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NEW ECLECTIC MEDICAL PRACTICE.

VOLUME I.

SECTION I.

GENERAL REMARKS ON FEVER AND INFLAMMATION.

I. FEVERS.

Synonyms.—Febris; Pyr; Pyretus; Pyrexia.

Definition.—A pathological condition characterized by elevation of temperature, acceleration of pulse, disturbance of circulation, arrest of secretion and excretion to a certain extent, disturbance of innervation, and loss of flesh.

General Classification.—Fevers have been divided into two general classes. In one the exciting cause is a zymosis, which provokes a primary constitutional disturbance (idiopathic), while in the other class the febrile action is secondary to a localized inflammation. But modern writers have discarded this classification, it being apparent from comparatively recent discoveries in bacteriology that the difference in many cases is more apparent than real. The modern classification would be into *Infectious Fevers* and *Non-infectious Fevers*, both classes including certain forms of idiopathic and symptomatic fever of the old classification. In the present work no attempt at strict classification will be made, as the subject is yet in an unsettled state, and infectious fevers will therefore be classed with acute infectious diseases.

The Detection of Fever.—The diagnostic symptom of fever is an elevation of the temperature of the body above the normal range (98.5° F., approximately). All other indications of fever, such as acceleration of pulse, disturbance of circulation, arrest of secretion and excretion to a certain extent, etc., may be present, and yet

without a disturbance of the temperature there will be no fever. However, in exceptional cases at particular periods it may be below instead of above the normal rate. The temperature-changes are detected by the use of the clinical thermometer.

Thermometry.—The ordinary thermometer consists of a glass tube marked with the degrees, and terminating at the lower extremity in a bulb which contains the bulk of the mercury, a portion of this rising into the tube and being further raised as warmth is applied to the bulb. A detached portion of mercury (the register) is separated from the main column and remains stationary after

Clinical Thermometer. (O. L. J.)

cooling, marking the highest point reached during each trial. This should be shaken down below 95° each time before using the instrument. Before and after using, the thermometer should be sterilized, especially in case of infectious diseases. In testing the temperature the bulb may be placed in the mouth, axilla, or rectum, as may be most convenient or seem most appropriate. In females the vagina may be resorted to instead. In order to insure full expansion of the mercury the bulb should remain in position for fully five minutes, care being taken to exclude the air by closing the lips when the bulb is under the tongue, or holding the arm snugly to the side when it is in the axilla. Delirious and comatose patients cannot be relied upon to retain the bulb in the mouth, and the axilla should then be resorted to. Packs or other dressings about the chest may then render it preferable to introduce the instrument into the rectum. Where the thermometer is a bugaboo to timorous children the rectum should be chosen. The temperature of the rectum is about one degree higher than that of the axilla.

Stages of Fever.—It has been a time-honored custom to name the three stages of fever the "cold," the "hot," and the "sweating" stage, respectively. These names have been abandoned by modern authors, however, as the sensation of chilliness is not always noticeable during the first stage, and the thermometer may demonstrate a marked rise of temperature even while the subject is experiencing the chilliness. Better terms, then, are: (1) the *stage of invasion*; (2) *stage of acme*, *fastigium*, or *stadium*; and (3) *stage of defervescence*, or *decline*. The stage of invasion in malarial fevers and others occurring in markedly malarious regions is usually signalized by a marked chill, or rigor. Outside of such influence, the chilly stage

of typhoid fever, scarlatina, measles, etc., may or may not be noticeable. During the stage of invasion the temperature is rising. During the stage of acme, fastigium, or stadium, it inclines to touch, repeatedly, the highest point; and during the stage of defervescence or decline, it is falling, either rapidly or by gradations, toward the normal point.

Termination of Fever.—Fever may terminate (1) by crisis, and (2) by lysis. Crisis is characterized by rapid and permanent decline of temperature; a decline of four or five degrees occurring in twenty-four hours, with sudden establishment of all the secretions and excretions, with disappearance of nervous symptoms and other discomfort. Lysis is attended by a gradual fall of temperature or by remitting gradations, the morbid symptoms subsiding gradually, the stage occupying several days or a week.

Febrile Remissions.—Prior to the general use of the fever-thermometer among physicians fevers were divided into two general classes, a division which the thermometer has shown to be faulty. The malarial fevers were classed as periodic; and enteric, typhus fever, and the exanthemata, were classed as continued. But the thermometer demonstrates that all fevers manifest a diurnal variation of temperature, the minimum being reached about 6 A. M., and the maximum about 6 P. M. Therefore, no fever is continued, in the sense suggested. However, certain forms are so nearly continuous in the maximum range that they are *classed* as *Continued Fevers*, though the arrangement is different from the old one. Typhus fever, scarlatina, and sometimes typhoid, in which the variation between the maximum and minimum temperature is only 1° to 1.5°, may be included in this division. In another division there are marked diurnal remissions, but the minimum never reaches the normal point during the fastigium, and only at or near the end of the decline. These are properly *Remittent Fevers.* Examples of this type are typhoid (usually), malarial remittent, and hectic. In still another class the temperature falls to normal or below that point, during the diurnal decline. Such are classed as *Intermittent Fevers.* The principal intermittent fevers are malarial intermittent, relapsing, hectic (occasionally, though usually remittent), and Charcot's intermittent (gall-stone fever). It will be observed that this classification cannot be very permanent, as severe cases of typhoid may hardly show diurnal variation of temperature at all, while mild cases, or those complicated with malaria, usually manifest marked remissions and exacerbations.

Causes of Fever.—The presence of microörganisms or their alkaloids (toxines) in the blood; local inflammations acting as

exciting causes; the products of fatty metabolism; and paralysis of the heat center.

Parasitic Origin of Fevers.—The past few years have thrown much new light upon the origin of infectious fevers. The microscope has opened a new era in the etiological phase of these diseases, and rendered obsolete doctrines respecting causation formerly promulgated. It is not impossible that changes may yet occur in views which are now almost universally accepted, but it hardly seems that so many observers can be mistaken as to the identity of the germs which are believed to be the exciting factors in several of the different forms of this class of diseases. If it be granted that the new doctrine is established in a single instance it cannot be doubted that all diseases of the same class—contagious and infectious fevers—will finally be traced to similar causes, and their specific germs pointed out and described.

It is pretty certain that infectious fevers depend for their origin and spread upon the propagation and transmission of specific microörganisms which are conveyed from sick to well, either directly or through fomites which serve to preserve and convey them, each disease depending upon its own particular germ, and never originating spontaneously. Some microörganisms have special indigenous habitats where they exist perpetually, to be widely disseminated under favoring influences, afterward dying out, except in their favorite haunts. Such is the character of cholera, which is indigenous to East India; yellow fever, indigenous to the Antilles; and typhus fever, indigenous to Northern Europe. Others, when they invade new territory, remain there permanently, on account of the power of the germs to resist external influences, hibernating at times and possibly gaining strength from filth and putrefaction. Examples of such are diphtheria and typhoid fever.

Infection, then, may be said to be the development, *within* the blood, from a transmitted germ or microörganism, of a colony of the same species, the excretions and secretions of which give rise to poisonous ptomaïnes (toxines), resulting in general sepsis of the fluids of the body, and consequent destructive action upon the blood and tissues. All organisms, during their activity, produce more or less waste or excrementitious material, which, in the case of the infectious microbe, constitutes the poisonous element. The virulence of the poison depends upon the individuality of the microörganism, and this accounts for the comparative severity of some infectious diseases and the mildness of others; while the condition of the system in different individuals, or their power of resistance to disease, may account for mild and severe cases in the same home at the

same time. A comparative severity and mildness of different epidemics is also observable, depending largely probably upon hygienic surroundings and atmospheric influences.

The human system may be infected with either *vegetable* parasites or microörganisms (bacteria), or minute *animal* organisms (hæmatozoa). Many of the bacteria are self-limiting in the human system, perishing either from lack of nourishing pabulum, from poisoning by their own toxines, by phagocytosis, or in some way not yet suggested, thus leaving the individual completely or partially protected from subsequent attacks. The hæmatozoa of Laveran, which are supposed to be the microörganisms of malaria, do not produce the severe septic effects caused by some of the bacteria, are not self-limiting, and if the disease they cause be arrested there is left a predisposition to a return of the malarial manifestation. It is possible that when the blood is once infected with malaria the principle may remain, in a more or less active state, perpetually.

The study of the microörganisms of disease belongs to bacteriology, and no more reference will be made to it in these pages than is necessary to discuss the practice of medicine intelligently. Really, Eclectics realized the importance of correcting septic processes, and had adapted specific agents to their correction, long before they were known to be caused by microörganisms; and we can do but little better, if any, in treatment now. But we have gained an important advantage in preventive medicine, and can now treat the subject with an intelligence and positiveness not possible with previous lack of information.

Symptoms of Fever.—Elevation of temperature, arrest of secretion and excretion, acceleration of the pulse and respiration, disturbance of the nervous system, coated tongue, traces of albumen in the urine, deposit of urates, loss of flesh and strength.

The *temperature* varies much in its maximum height in different cases, and in its average maximum height in different forms. Some fevers are characterized by an unusally high maximum temperature, and the moderate rate is the exception. Such are typhoid fever, scarlatina, typhus and relapsing fevers. Others are fevers of low maximum temperature, as a rule, such as rubella, cerebro-spinal fever, and measles. Some protracted fevers which terminate by slow lysis are marked near the close by subnormal temperature, the thermometer registering below 97.5° F. during the morning remissions. The subnormal temperature is rather common to the convalescent period of typhoid fever and pneumonia; also in collapse from shock, hemorrhage, heart failure; or perforation of visceral walls, as of the bowel in typhoid fever, the lung in phthisis, or the stomach

in round ulcer. The temperature may be subnormal in certain chronic diseases, such as diabetes, cancer, and chronic cardiac, cerebral, and spinal affections. The temperature of cholera is remarkably subnormal, it frequently remaining at 90°—85° for several days.

The temperature of some fevers follows a pretty constant course, in the majority of cases; thus, the temperature of typhoid fever runs a typical course usually, and so does that of typhus, relapsing, malarial, and other fevers. Others are notoriously irregular in this respect—cerebro-spinal fever and diphtheria, for example. *Complications* are marked by sudden changes of temperature, thus: The advent of nephritis or inflammation of other important organ in scarlatina, diphtheria, etc., is announced by a rapid rise of temperature; intestinal hemorrhage in typhoid fever is characterized by abrupt decline of temperature; etc. In all severe cases of protracted fever, frequent use of the thermometer will enable the practitioner to detect complications much earlier than he otherwise would, and will prepare him for proper therapeutic adaptation at an early stage, when his change of treatment may be of benefit.

Wunderlich has made the following classification of the temperature of fever, which is worthy of record:

1. Subfebrile; temperature 99.5°—100.4°.
2. Slightly febrile; temperature 100.4°—101.3°.
3. Moderately febrile; temperature 101.3°—103.1°.
4. Decidedly febrile; temperature 103.1°—104°.
5. Highly febrile; temperature 103.1° in the morning and above 104.9° in the evening.
6. Hyperpyretic; above 106°.

A moderately elevated temperature without remission is more to be feared than one that is much higher in its maximum but which declines markedly each twenty-four hours.

The *pulse* is increased in frequency in most fevers, though during the stage of calm in yellow fever it becomes remarkably slow, being reduced to forty or fifty per minute; and in malignant forms of malarial fever it may be abnormally slow. The pulse is easily disturbed by slight causes in early childhood, and is then not of much importance as a symptom, either in diagnosis or prognosis. The *quality* of the pulse is as important as its frequency; the small pulse of debility, the strong pulse of sthenia, the full, hard pulse of obstruction, the full, bounding pulse of sthenia with arterial relaxation, the oppressed pulse of capillary congestion, the sharp, wiry pulse of nervous irritation, the feeble, fluttering pulse of impending dissolution or cardiac debility, each carries its suggestion to the observant practitioner.

Disturbance of the *nervous* system may vary from slight restlessness to extremely violent delirium, in which it may be necessary to employ force to prevent the patient from getting out of bed. Two kinds or qualities of nervous disturbance are observable in different cases; viz., that of active irritability, and that of oppression or drowsiness. In one there is irritation, and in the other there is intoxication, from the disturbing toxine. The active symptoms are most liable to appear early in the course of a fever, and the second later on, though either may be marked from the beginning, while in other cases the nervous symptoms may not be at all prominent at any time.

The *tongue* furnishes important symptoms in many cases of fever regarding diagnosis, prognosis, and treatment. The tongue of scarlatina is peculiar and almost diagnostic, that of gastric irritation unmistakable, and other morbid conditions are just as certainly shown by the tongue. We are enabled to select many remedies with tolerable certainty, and thus meet varying conditions of disease with a readiness not otherwise possible. Special notice will be given this subject under the head of treatment.

Tissue Changes Resulting from Fever.—High and long-continued fever results in considerable change in the quality of the tissues, the amount of fever bearing an important relation to the extent of morbid change. After high and prolonged fever the different organs are more or less swollen, opaque, and friable. Evidence of recent circulatory disturbance is furnished by the injected vessels and general œdema. Microscopic appearances indicate marked alteration of histological elements; the cellular elements are increased in size and their protoplasm has become granular, obscuring the nucleus. The granular condition is due to the presence of albumen and fatty particles. The tissue-changes occur most markedly in pyæmia, erysipelas, typhus, typhoid, and other infectious fevers, and in acute rheumatism. Alterations may be so extreme as to amount to necrotic changes, such as those in the tissues of the liver in yellow fever, though this is probably due largely to the specific character of the disease. The organs in which pyrexial changes are most observable are the liver, the heart, the kidneys, the muscles, and the lungs.

General Treatment of Fever.—*Rest* in bed is the first essential, and the earlier the patient gives up exertion and affords every assistance possible in this way the better are his chances of an early recovery. In many cases the early symptoms are so urgent that the patient succumbs at once and goes to bed; but in other cases, such as typhoid fever for example, the onset may be so insidious that he may remain about and on his feet until his recuperative

energies are too nearly exhausted to assist him through the trying ordeal which follows. But physical rest is not the only essential here. Where there is the least tendency to nervous irritability, the room should be darkened and all noise strictly prohibited. Talking must not be allowed in the sick room; even whispering must be prohibited.

Ventilation is also highly important in the treatment of fevers, especially those of an infectious character. The emanations from subjects affected with diphtheria, typhoid, typhus, or yellow fever, in an unventilated room, are always additional elements of danger. It has been observed in epidemics of both typhus and yellow fever that patients who have been carried out of hospital wards in an apparently hopeless condition into the open air have revived, and in some cases gone on to complete recovery; and it is a favorite practice with those of extensive experience with these diseases to treat them in tents or open barracks. But neglect of ventilation must not be permitted in any infectious disease, though in such an instance as measles cold air is not allowable, on account of its irritating influence upon the sensitive respiratory membrane. The temperature of a fever apartment should ordinarily be about 60° F., though no rule can be laid down to supplant the discretion of the physician in individual cases. In each case the temperature should be maintained at about the same degree of warmth throughout the disease.

The *diet* in fevers should, as a rule, be liquid or semi-solid. In most fevers morbid changes occur in the mucosa of the alimentary canal which incapacitate this tract for the performance of its usual functions. As a liquid diet is more easily digested and assimilated, it must consequently be the appropriate form for use. In case of typhoid fever the use of solid food is absolutely dangerous, hazarding perforation of the weakened intestinal wall; while in diphtheria and scarlatina a liquid diet is more readily swallowed. Doubtless, the natural efforts required for the digestion of solid food disturb the heat center indirectly, and so occasion more or less rise in temperature. Therefore, in all protracted fevers especially, a liquid diet is the only admissible form of food for use. But it is not alone requisite that the diet shall be liquid. *Quality* must be considered. It was once believed that beef tea was all the food necessary for a fever patient, but it is now generally conceded that one would starve in time if fed upon nothing but this article. It has been asserted, with good reason, that it contains no more nourishment than urine. Representing, as it does, the products of a destructive metabolism, it hardly seems capable of even exerting the stimulant influence attributed to it. *Nourishment* is an important qualification of liquid

foods, and we find this varying in amount in different kinds. *Milk* is a leading article in this line, but its use is attended by the objection that the curd which forms after it becomes acidulated in the stomach may become a firm and resisting mass, difficult to dispose of. This objection, however, may be obviated by adding from one to two ounces of lime-water to each pint of milk before using. In these days *prepared foods* have done away with many of the problems of old in the feeding of fevers. Among those to be especially commended are malted milk, lactated food, and Mellin's food. I have fed many cases of typhoid fever on Mellin's food throughout, with most excellent satisfaction. Vegetable broths, soups, and gruels are excellent alternates when the animal foods become objectionable; and some of them are very nourishing, such for instance as rice water, bean soup, and oat and cornmeal gruels, though these should be carefully strained when used in enteric fever. Food should be given often and in small quantities during the active stages of protracted fevers, the same regularity and promptitude being observed as in the administration of medicine.

In most cases of fever the fauces are dry and *thirst* is an urgent symptom. There is no objection to the free use of water in such cases, unless there be gastric irritability with rejection of fluids as soon as swallowed; in which case the stomach must be given complete rest and proper medication for a time, while the thirst is palliated by packing the epigastric region with a folded towel which has been wrung out of cold water, or by using a rectal injection of cold water, to be retained. Liquid diet answers well here, sustaining as well as relieving thirst, and whey, barley-water, toast-water, koumiss, lemonade sweetened with maltine or grape-sugar, tamarind-water, and many other articles may be selected to satisfy the taste, or to conform to other requirements. Bits of ice may be held in the mouth and allowed to dissolve, but this must not be carried too far, for fear of embarrassing recuperative processes by chilling the stomach.

In all protracted fevers the danger of permitting the patient to remain continually in one position should not be overlooked. Hypostatic congestion of the lungs is almost certain to follow where a person is allowed to lie upon the back or in any other fixed position for a long time, and fatal sequelæ are very liable to follow getting up, from the pulmonary complication. Bed sores are always a menace, and liability to them is much increased when pressure is made constantly upon one part for protracted periods. It is the duty of the physician to know these facts, and to instruct attendants to turn the patient every three or four hours, so that he may lie a portion of the time on each side and a portion of the time on the back.

The *medicinal treatment* of fevers will vary considerably in different varieties, and also in different cases of the same character. In brief, there can be no fixed course to pursue, but each case must be individualized, and treated as a separate proposition. It may seem from this statement that the subject must be a very complicated one, then, but this is not so, for a proper understanding of the principles involved reduces the problem to a few simple propositions.

While remote causes of fever have been noticed, it is to be remembered that toxines generated by the presence of parasites or other impurities are the immediate elements to be considered. Though the foreign element may have been provocative of the condition, nature has apparently provided the means for its final elimination, if, in the meantime, the zymotic action has not been so extensive as to destroy the life of the patient. It appears to many that efforts of therapeutists toward the destruction of disease-producing germs after they have entered the circulation will always prove futile, as many germs are capable of resisting the action of drugs powerful enough to destroy human life when swallowed. In the rational treatment of fevers, then, we will limit ourselves to the management of the poisonous ptomaïnes generated, and their effects.

One of the great dangers in fever lies in the high temperature which attends many cases. This interferes with secretion and excretion, and encourages degeneration of tissue, wasting, and loss of strength, as well as favoring fatal changes in such delicate and sensitive organs as the brain, lungs, and other vital parts. We will strive, then, from the commencement of treatment to lower the maximum temperature toward the normal point by every *safe* and rational means. The popular plan for the accomplishment of this purpose among the most successful class of practitioners is the use of the **special sedatives,** in minute and frequently repeated doses. The recognized special sedatives are: aconite, belladonna, gelsemium, jaborandi, veratrum, and rhus tox. Though powerful depressants in large doses, these remedies exert a remarkably calmative effect upon the circulation in many cases of fever, without depressing, when administered in minute doses, and repeated as often as every hour, day after day. Not only are the force and rapidity of the circulation and frequency of the heart's action diminished, but nervous erethism is calmed, and secretion promoted. Each one manifests decided peculiarities which adapt it to special cases or conditions. These conditions are suggested by the character of the pulse usually, though other symptoms may assist in the selection of the correct agent. The following hints are submitted:

Aconite is the remedy for the ordinary fever of debility in middle life, in children, and elderly adults. The characteristic pulse is small and rapid, but distinct and regular (not wiry). It is applicable to infectious as well as non-infectious fevers, while it assists in controlling local inflammation, especially that of mucous membranes. It is calming to conditions of nervous excitability, though not as useful as rhus tox. where this symptom is marked. It promotes normal secretion, especially from the skin and mucous membranes; and though it quiets excitement of the circulatory organs, it doubtless improves their normal energy at the same time. In using, for an adult, add five or eight drops of Lloyd's or Worden's aconite to four ounces of water, and administer a teaspoonful every hour.

Belladonna furnishes us with two peculiarities of action, depending upon the method of administration. When half a drachm or a drachm of the third decimal dilution is added to four ounces of water, and a teaspoonful given every hour, it is applicable to debilitated conditions marked by furious delirium. In this case the eyes are wild, the face is flushed bright red, the mind abnormally active and aggressive, while the pulse is small, feeble, and oppressed, and the patient markedly prostrated. In the other case we obtain its effects from more material doses. Adding five or ten drops of the specific medicine or a green plant tincture to four ounces of water we have a remedy for febrile conditions attended by feeble capillary circulation, suggested by coldness of the extremities, feeble, oppressed pulse, inelastic tissues, dullness or drowsiness, sensation of swimming in the head, dilatation of the pupil, muttering delirium, etc.

Gelsemium is the remedy for febrile conditions in sthenic subjects, at least those in which prostration is not a marked feature. It controls vascular excitement, promotes secretion, relaxes spasm, and alleviates pain. The typical indications for it are a full bounding pulse, flushed countenance, bright eyes, and contracted pupils. The delirium of gelsemium is of active character. In using for its sedative effect, from twenty to thirty drops of a saturated tincture of the fresh root should be added to four ounces of water, and a teaspoonful of this administered every hour.

Jaborandi is another sedative for sthenic conditions. It controls vascular and cardiac excitement, promotes secretion, especially from the skin, and alleviates muscular pain. Its cooling influence upon the skin imparts a grateful sensation to the fever patient, and modifies the exalted temperature shortly after its use is begun. In order that the agent may be reliable it must be prepared from the fresh crude article; the ordinary fluid extracts of the market being

comparatively worthless for sedative purposes. When using, add from one to three fluidrachms to four ounces of water, and give a teaspoonful every hour. Full, strong, hard pulse, with dry skin and severe muscular pain is a special indication for its use.

Veratrum is also a remedy for sthenic febrile conditions. It sedates vascular excitement of the general circulation, lowers an exalted temperature, and promotes general secretion. It fills much the same place as jaborandi, though it is not as satisfactory. Its special indication is a full hard pulse, with elevation of temperature. It is contraindicated where gastric irritation is present. In using, add fifteen or twenty drops of a reliable drug to four ounces of water, and give a teaspoonful every hour.

Ferric Phos. is Schüssler's remedy for fever, and it often proves reliable, its searching effects sometimes becoming appreciable after the special sedatives have failed. It is especially adapted to the treatment of symptomatic fevers before plastic exudation begins in the inflamed part. In using, add three grains of the 3x trituration to four ounces of water, and give a teaspoonful every hour.

Potassium Chloride is the remedy for symptomatic fever after plastic exudation has begun, as it promotes rapid removal of the exudate by absorption, thus preventing obstructive organization and destructive changes. In using, add five grains of the 3x trituration to four ounces of water, and order a teaspoonful every hour.

Adjuvants may often be employed with advantage to aid the influence of the special sedatives. Prominent among these are the *vapor bath, cold, tepid and hot packs* and *baths,* and various *enemata.*

But though the special sedatives answer an excellent purpose in the treatment of non-infectious and malarial fevers, as well as in some other mild infectious fevers, such as roseola, measles, mumps, and chicken-pox, there are others where there are such rapid destructive and septic changes in the blood and tissues, that they are almost or wholly inefficient. In typhoid fever, typhus fever, yellow fever and diphtheria, where general necrotic changes are pronounced, little satisfactory use can be made of them. We must then depend upon the **antiseptic sedatives**—remedies which combine antiseptic, antinecrotic and stimulating properties with those of a special sedative. Prominent in this class are echinacea, lachesis, baptisia, and salicylate of ammonium.

Echinacea is not only an arterial sedative,—not quite as markedly so as the special sedatives, probably,—but it controls necrotic tendencies in the blood and tissues, both when used locally and when taken internally. It seems to be an organizer, improving the vitality of the circulating fluids and tissues, fortifying them against septic and

necrotic changes. In all febrile conditions where septic states tending to necrosis of tissue are common, as well as where they are actually present, it commands the leading place as a remedy. The pathology of the disease in question will afford the indications for it—breaking down of the blood corpuscles with destruction of the fibrin element, and granular degeneration of the fixed cells, with localized necrosis of the soft tissues. This we find, to great or less extent, in all severe infectious diseases. The average dose for an adult is ten drops of a saturated tincture of the recent plant, or its equivalent, repeated every hour.

Lachesis comes nearest echinacea in its power over necrotic conditions occurring in febrile diseases. It improves the power of the heart when this organ is laboring under the depressing influence of toxines, and stimulates the organs supplied by the pneumogastric nerve generally. It is especially indicated in infectious diseases attended by feeble, tremulous heart induced by toxic causes. In fevers of low form, where there is marked prostration with phagedenic tendencies of the tissues, as in malignant scarlatina, diphtheria, hemorrhagic variola, etc., it is perhaps our best recourse, especially where cardiac failure portends. The 6x trituration may be administered in two-grain doses every two or three hours in such cases.

Baptisia is recommended in a class of cases similar to those in which echinacea is so efficacious. It acts as a sedative and stimulant in typhoid conditions, and controls to some extent necrotic tendencies, sloughing of tissue in the intestine in typhoid fever, the throat in scarlatina and diphtheria, etc., responding to its action. Duskiness of tissue, prune-juice discharges and low muttering delirium indicate it. We once thought that we possessed the acme of treatment in such conditions when provided with this remedy, but there is little doubt that echinacea far excels it in any case where it is adapted. From two to ten drops of the saturated tincture of the fresh root or its equivalent, may be administered every hour or two in cases requiring it.

Salicylate of ammonium is an antiseptic sedative of rare virtue where a persistently high temperature renders a case of infectious fever especially serious. When other remedies fail in such cases we may safely depend upon this agent to reduce the temperature, and it is an antiseptic of excellent service at the same time. Full directions for preparing, dose, etc., can be found in "Dynamical Therapeutics."

Another class of remedies, which I shall here term **correctives**, fulfills important indications in fever on many occasions. The action of these remedies is chemico-vital in nature perhaps, the result tending toward the correction of excessive acidity or alkalinity.

They can hardly be considered as antiseptics, but they correct conditions which materially interfere with the proper action of remedies generally. Three important remedies of this class are sulphite of sodium, sulphurous acid, and hydrochloric acid.

Sulphite of sodium is a corrective where there is a heavily loaded tongue, presenting a pasty-white appearance, with pallid mucous membrane. This indicates excessive alkalinity of the system, and a salt of sodium is the natural corrective. Before we can reasonably expect other remedies to produce their ordinary effects this must be corrected. A few days of sulphite of sodium will accomplish this, and all other aggravated symptoms will be correspondingly modified. The sulphite may be administered in one- or two-grain doses every three or four hours during the day, until the morbid condition of the tongue calling for it has disappeared, which will usually be within three or four days. It is advisable to administer the remedy in capsules, where swallowing is readily accomplished.

Sulphurous acid is a corrective where the tongue is coated brown, with dark sordes on the teeth and lips, or even without these accompaniments. Under this coating the mucous membrane is darker red than is natural, it usually being uncovered at the sides and tip. Low forms of fever often present us with this condition, and sulphurous acid is then an appropriate remedy. Twenty drops of the acid, well diluted in water, may be given every three or four hours.

Hydrochloric acid is the corrective where the tongue is red, slick, and shining, presenting a beefsteak appearance. Probably there is a lack of acid in the system in these cases, as acids seem to help lagging cases immediately where the indication is marked. Fifteen or twenty drops of dilute hydrochloric acid, in syrup, may be administered in such cases every four hours, to prepare the way for other remedies.

The subject of *antiperiodics* demands a little space. There are certain fevers characterized by marked periodicity which seems to be the leading feature of the attack, and which must be interrupted before much progress can be made toward a cure. There are those who even argue that the cure is completed when the periodicity is interrupted; but this is a mistaken idea, for the physician who expects to *cure* malarial attacks with quinine or other antiperiodics will find himself disappointed in a majority of the cases treated. Quinine and other antiperiodics—notably quinine—interrupt periodicity, but do not remove the *materies morbi* from the system, and the periodical manifestation returns after a cycle, or multiple of cycle, of seven days.

While antiperiodics, then, are important remedies to interrupt marked periodicity where the cause arises from malarial influence,

curative effects can not be expected from them usually, and rational means applied to the morbid conditions existing must follow their use if permanent benefit is to be insured. *Quinine* is the ideal antiperiodic, and the one which is usually employed. But, unfortunately, it is not always a remedy which can be administered without objectionable results. When administered to certain patients it produces ringing in the ears, nervous irritability, and even delirium, aggravating already-existing evils instead of benefiting them. The proper time for the administration of quinia is during the intermission or remission of periodical diseases, and the system should be prepared for its use by the previous administration of such remedies as render the tongue moist and cleaning, the pulse soft and open, and place the nervous system in a condition of tolerance. This can frequently be accomplished by the use of the special sedatives, selected as already suggested. Quinine is of little use in the treatment of infectious fevers caused by bacteria. It seems to disturb the patient and aggravate the symptoms, in the majority of cases. Its principal place is in the treatment of malarial conditions, and here we only rely upon it to interrupt the periodicity.

There are other antiperiodics which sometimes excel quinine in certain respects, and are worthy of notice in this place:

Arseniate of quinia, 3x trituration, is not usually as active as quinine, but it is sometimes more efficacious, and is less unpleasant for children and sensitive adults. It is better adapted for steady administration in chronic cases, as it does not leave behind the objectionable effects of quinine upon prolonged use.

Alstonia should also be recollected where stubborn cases of periodicity are met, for it will repay careful study and trial. It is recommended in malarial cachexia where the tongue is coated and the urine loaded with phosphates. It seems more permanent in its effects than quinine, though slower in action.

Faradism, the "tonic treatment" being employed, is not to be forgotten in the treatment of chronic periodicity. When assisted by *cabinet vapor baths* it is almost invincible.

The use of stimulating tonics during convalescence should have gone out of fashion long ago. Such drugs as strychnia, nux vomica, calisaya, quinia, etc., tend to derange digestion, set the nervous system on edge, and thus oppose recuperative processes. Such agents may be of some service in preventing the return of malarial attacks, but are usually objectionable during convalescence from other infectious fevers.

Undue muscular exertion should be avoided during convalescence, the patient being prohibited from rising from bed before the weak-

ened condition of the heart-muscle and degenerated tissues generally has been corrected. The food should be selected with care during this time, and adapted to the particular condition which may have been brought about by the morbid action preceding. After typhoid fever and certain other diseases the alimentary mucous membrane is in a debilitated condition for several weeks, and the return to ordinary diet should be gradual, the food being selected with due regard to this fact. After all cases of protracted infectious disease the digestive apparatus is weakened, and recuperates slowly, and there is great danger in the early use of crude and indigestible food soon after convalescence. Diphtheria is an affection in which collapse and death have followed injudicious feeding early after convalescence; and this danger arises in every case of severe infectious disease.

The abuse of *opiates* in fevers in past years has been a matter of common record. Even our old Eclectics cannot plead innocence in this direction, as their favorite remedy for febrile conditions was formerly the diaphoretic powder, containing enough opium to disturb the nervous system unpleasantly, in many instances. There are few febrile conditions where opium or its alkaloids can be used successfully Doubtless, in many cases, life has been sacrificed by the stupid administration of this drug. The practice of old, with its crudities, could recognize but one remedy for restlessness and pain, and that was some form of opium; and as such symptoms were common, it entered largely into the ordinary treatment of fevers. It excites the brain and nervous centers generally, arrests secretion and paralyzes function, with resulting debility after the narcotic effect has passed off. Delirium, increase of pyrexia, dryness of the tongue and skin, with diminished urinary excretion, follow its use, while enough of the drug to cause slumber in the wakeful and restless fever-subject is liable to produce profound prostration. Its occasional use may be allowable, but its objectionable features as a drug for common use should be fully realized. Unfortunately, it has been superseded, to some extent, by a class of drugs—the coal-tar products—which may leave behind extreme prostration and cardiac debility, from which recovery is slow and difficult. Our materia medica contains numerous safe resources for the relief of such symptoms as those for which these drugs are prescribed in fevers, as every diligent and observing practitioner must know. Opium may be administered cautiously where the skin is not dry, where the pulse is soft and open (not hard or wiry), where the tongue is moist and normal in shape (not dry and contracted, nor reddened and pointed at the tip), and where the nervous system is

not markedly disturbed. Of course there are extreme cases where all indications may be disregarded and lethal doses of opiates administered, these being hopeless, and attended by excruciating pain.

Muscular pain, where opiates were once employed so extensively, and where the coal-tar derivatives are now administered too frequently, is a common symptom of many fevers. When rheumatoid in character, as it often seems, it may usually be alleviated by the use of organic remedies which leave no ill-effect behind. Among these may be mentioned rhamnus californica, especially valuable where a laxative effect is desirable; cimicifuga; caulophyllum; bryonia, when the pain is principally about the thorax. Salicylic acid from wintergreen, and salicylate of sodium may be of service. And sometimes phenacetin—one of the least objectionable of the coal-tar products—will be found to answer the best purpose. However, it should be recollected that the muscular pains which attend cerebro-spinal fever and some other affections depend upon localized irritation of nervous structure, which must be removed before muscular pain can be controlled with these remedies. Here, the local nervous lesion will demand first attention.

Cathartics, which were once supposed to exert an important influence in the treatment af fevers, are used but little by modern practitioners. The idea that febrile excitement can be materially lessened by their action now receives little credence. There is no doubt that such drugs disturb and irritate the intestinal mucosa, interfering with digestion and assimilation; and when it is recollected that the alimentary canal is not an excretory apparatus, and that there is a tendency toward structural degeneration here in most fevers, the common use of cathartics in their treatment seems extremely irrational.

II. INFLAMMATION.

Synonyms.—Phlegmone; Phlogosis; Phlegmasia; etc.

Definition.—A localized disturbance of cellular and vascular function, of destructive tendency, characterized by hyperæmia, exudation of blood- and tissue-elements, and migration of leucocytes, attended by pain, heat, redness, swelling and impairment of function in the part affected.

Etiology.—The causes of inflammation are local irritants, which may be divided into two general classes, viz., simple and infectious. Simple causes are those which are non-infectious. They comprise mechanical injuries; chemical effects, such as the action of caustics; extreme congestion due to sudden arrest of secretion; etc. Infectious causes comprise numerous varieties of microörganisms, some

of which have been separated, cultivated and studied, and some of which exist at present only in imagination. At least, their presence has not yet been satisfactorily demonstrated. However, there are extremists who assert that all true inflammatory action depends upon the presence of microörganisms in the affected tissues.

Infective inflammation may probably arise either from the direct action of the microbes upon the tissues, from the irritation of their toxines, or, as is usually the case doubtless, from a combination of these influences.

Pathology.—The pathology of inflammation is so complicated that a separate study of some of its essential features will be most likely to convey a clear conception of the nature of the disease. We will therefore consider in the beginning the principal histological elements concerned.

HISTOLOGICAL ELEMENTS INVOLVED.

Blood-vessels. The principal vascular changes of inflammation occur in the capillaries, though the minute veins participate. Through the walls of these vessels the blood-elements make thier escape, and through them the exudate largely enters the circulation after the inflammatory action has subsided. The walls of the capillaries consist of a single layer of nucleated endothelial cells, united by an interstitial cement-substance. Inflammatory action disintegrates the interstitial substance at various points, and openings (stomata) are left between the edges of the cells, through which the inflammatory exudation makes its escape. Mechanical and chemical influences doubtless conspire to bring about this condition. The engorgement of the vessels gives rise to distension and intravascular pressure, and the large volume of blood in the part increases the heat, this combination of forces soon resulting in defective points in the cement-substance, where stomata afterwards appear.

CAPILLARIES, MAGNIFIED.

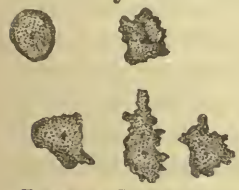
There seems to be an excitement in even the most minute blood-vessels which suggests a nervous stimulus. The arterioles pulsate tumultuously, and the entire inflamed part is filled with a throbbing sensation, probably due to vascular excitement and nervous erethism. The developmental and nutritional properties of these organs are also evidently disturbed, as evidenced by the fact that vascular tufts may be developed during inflammation in such non-vascular structures as the cornea; and new capillary loops are occasionally put forth exuberantly in other tissues.

The Blood-corpuscles. Three kinds of blood-corpuscles exist, and all are concerned in inflammatory action. These are (1) the white

corpuscles, (2) the red corpuscles, and (3) the blood-plaques or third corpuscles. The accompanying diagram is suggestive of the relationship which these sustain to the circulation. The globular bodies near the wall of the vessel are white corpuscles, the oblong figures accompanying them suggest the third corpuscles, though these are not readily visible in circulating blood, and the dark central band of disks the red corpuscles. The white corpuscles, being of lower specific gravity than the red, are crowded out of the center of the current, and move along the periphery.

Blood-corpuscles.

The *white corpuscles* take the most active part in the process of inflammation. They possess the power of spontaneous motion, and leave the capillaries and veins at an early period, migrating by amœboid motion through the connective tissue, there being adapted to a variety of functions. Leucocytes are continually undergoing change of form, their locomotion being due to this property. Infolding processes enable them to take up floating particles and inclose them in their substance. How these foreign particles are afterward disposed of does not seem very clear. Possibly pathogenic microbes are aimlessly distributed through the tissues and blood-vessels, other débris being scattered along in much the promiscuous manner in which it is taken up. Doubtless the leucocytes exert a solvent or digestive influence upon certain substances incorporated. Leucocytes should not be confounded with *embryonal connective-tissue cells*, which are at first detached, possess the power of amœboid motion, and resemble them very closely morphologically, but differ, from the fact that they may become fixed tissue-cells at a later period, and assist in the repair of damaged structure, or play a very mischievous part in interstitial inflammation.



Forms of a Single Leucocyte Observed within Ten Minutes.

A leucocyte consists of a hyaline mass contained in the meshes of a reticulum of protoplasmic fibers and containing a nucleus. The nucleus and reticulum constitute important parts of the structure, and are broken up during degeneration into a pus-corpuscle.

The destination of a migrating leucocyte during inflammation is uncertain. If the inflammatory action be slight and terminate in resolution, it usually assists in clearing up the affected area and then returns to the circulation, either directly, through a stoma, or by way of the lymphatics. Where the inflammatory action is more severe and there is considerable destruction of tissue, its substance may contribute to the growth of embryonal tissue-cells (after deliquescence). In another instance it may be converted into a pus-corpuscle.

The *red corpuscles* are too well known to require description. Possessing no power of spontaneous motion, their passage through the walls of the blood-vessels is wholly a passive process, due to a damaged condition of the vascular walls and engorgement of the vessels. The number of red corpuscles outside the blood-vessels in an inflamed area will suggest a relative amount of disturbance of this character. Being intimately connected with the production of animal heat, it is more than probable that the abnormal elevation of temperature observed in inflamed areas is due to the increased number of red corpuscles present. The same observation applies to the redness observed in an inflamed part, increased quantity of arterial blood imparting the heightened color. Being incapable of amœboid motion, the red corpuscles are not such common carriers of pathogenic germs as white corpuscles, and as they do not return to the general circulation after diapedesis, they probably exert little influence in the spread of infection. After the inflammation has subsided they become broken up, and the detritus is absorbed.

The *third corpuscles* are small colorless spheres or granules, twenty times as numerous as the red corpuscles. They probably represent the fibrin-element of the blood; and it is likely that they exude from the vessels during inflammation and constitute the principal bulk of the fibrin found in inflamed tissues.

Fixed-tissue Cells. The fixed-tissue cells participate in the histological changes of inflammation. Where the inflammatory action is severe, death of a community of these cells may occur in the center of the affected area, a mass of necrosed tissue marking the site. This is observed upon a small scale in a common boil (furunculus), and upon a larger, in phlegmonous inflammation. The direct cause of this necrotic influence is not yet satisfactorily settled. Radical believers in the doctrine that all clinical inflammation is microbic in origin, ascribe the condition to either the direct action of microörganisms, or to powerful toxines generated by them, except where starvation of the cells occurs from strangulation of their blood-supply. But, as it is generally admitted that chemico-vital influences are largely concerned in the destruction of the interstitial cement in the capillaries whereby these vessels become permeable by the blood, it might not be unreasonable to ascribe destruction of fixed-tissue cells to a similar influence, occasionally, at least. In chronic inflammation, instead of destruction of fixed-tissue cells, as in acute inflammation, there is proliferation of these elements, these furnishing the bulk of the inflammatory product, instead of exudation. This process, though not immediately destructive, may give rise to extremely serious results, by impairing the

functions of such vital organs as the liver and lungs. In interstitial hepatitis (cirrhosis) and interstitial pneumonia, proliferation of connective-tissue cells is almost invariably attended, sooner or later, by fatal results.

Exudation. The exudation which occurs in inflammation consists of solid and liquid parts. The solid parts are represented by the blood-corpuscles, which have already been considered. The liquid part, which consists of blood-plasma, is termed the *inflammatory transudation.*

This accompanies the corpuscles in their passage through the openings in the vessels caused by the damage to the vascular walls, its escape being due to the *vis a tergo* and the porosity of the vascular structures. The swelling which attends acute inflammation is largely due to the inflammatory transudation, the amount in the affected tissues determining the extent of the tumefaction. The œdema which attends certain inflammatory conditions is the result of excessive transudation. This is likely to occur when parts freely supplied with connective tissue are involved, such as the deep muscular tissues, the lungs, the eyelids, scrotum, etc. Where the parts are firm and there is little connective tissue, there is but small amount of inflammatory transudation. After inflammation subsides, the transudation is removed by absorption unless suppuration occur, in which case it becomes the pus-serum.

Where deep-seated inflammation occurs, the exudation is poured out into the cellullar tissue and lymph spaces, where the various changes already described are carried on. This may be *interstitial*, or *parenchymatous* inflammation. When acute, the exudation is increased by the addition of liquid elements from the tissue-cells. In *suppurative* inflammation, either a part or the whole of the exudation is transformed into pus. The blood-corpuscles, as well as many of the fixed-tissue cells, become pus-corpuscles. *Hemorrhagic* inflammation is characterized by the presence of an excessive number of red corpuscles in the exudation. In most cases of inflammation a few red corpuscles escape from the blood-vessels, but in this instance enough are poured out to constitute actual hemorrhage, the exudation presenting a reddened appearance suggestive of the condition. Such cases are attended either by serious local or general lesions. A very high grade of inflammatory action may prove sufficiently destructive to the blood-vessels to bring about this result. Depravity of tissue from previous disease, or serious obstruction to the general circulation, as in valvular disease of the heart, chronic nephritis, hepatic cirrhosis, etc., dispose to such condition, as well as syphilitic and erysipelatous complications.

Usually, in inflammation of *serous membranes*, the exudation is

poured out upon the surface, and the leucocytes and third corpuscles are here destroyed, the combination of the fibrin ferment, débris from the leucocytes, and blood-plasma forming fibrin, constituting a thick layer of coagulable material, which becomes firmly welded to the serous surface by means of capillary blood-vessels and granulations, these sprouting up and growing into it. This forms a nidus or hot-bed for the proliferation of connective-tissue cells, and as the endothelium participates actively in the inflammatory changes, perforations occur, through which embryonal connective-tissue cells wander, to undergo rapid multiplication and organization, until the exudation is entirely removed and replaced by connective tissue. If opposing surfaces—such as the reflections of the pleura or peritoneum—are involved, entire serous sacs may thus be obliterated, a growth of new connective tissue completely bridging the chasm. If, on the other hand, the endothelial cells remain intact, no embryonal connective-tissue cells are liberated, and the exudation is absorbed, leaving the serous surface free. If a serous inflammation be severe enough to result in suppuration, the leucocytes and embryonal cells become converted into pus-corpuscles, and collections of pus, such as empyema, pyocardium, and purulent peritonitis, result. Where the transudation is largely in excess of the corpuscular elements of the exudation, the inflammation runs a sub-acute course, and results in the accumulation of a considerable quantity of serous fluid in the cavity.

When *mucous membranes* are involved, the mucous follicles secrete profusely in most instances, flushing away the exudation which is thrown out upon the surface, and constituting *catarrhal inflammation*. At a later stage, the leucocytes and embryonal cells become converted into pus-corpuscles, and these mix with the mucus to form a *muco-purulent* discharge. In *croupous inflammation*, the fibrin-element predominates, and a coagulum which resists the flushing influence of the catarrhal discharge is formed, and this coagulum becomes more or less firmly attached to the mucous-epithelium, the cells of which undergo a process of necrosis. Thus is formed the pseudo- or false-membrane. When the necrosis of the mucous membrane is only slight, the secretion of the mucous follicles beneath the exudation may suffice to lift it away; in more severe cases,—as in diphtheria, for example,—the entire mucous membrane is involved in the necrosis, and the separation involves a slower process of sloughing.

In catarrhal inflammation of mucous membranes, thickening may occur from inflammatory induration of the submucous tissue following organization of the exudation. The large amount of fibrous material thus developed causes contraction, which, in tubular organs, may amount to permanent *stricture*.

PHENOMENAL HISTOLOGICAL CHANGES OCCURRING DURING INFLAMMATION.

When inflammation occurs in thin, transparent, vascular structures, such as the web of a frog's foot, tongue, mesentery, bladder, etc., and the part is properly placed under a microscope of the requisite power, some of the histological phenomena presented may be seen and studied. After the part has been properly placed and secured, inflammation is excited by the action of some irritant, usually a caustic, like the point of a red-hot needle, croton oil, nitrate of silver, or some similar irritant to animal tissues, and an excitation in the minute bloodvessels in an area surrounding this point is soon observable.

If the normal condition of the blood-vessels is carefully noted before the irritant is applied, a striking change will be apparent after the inflammatory action has become fully developed. In the normal condition, the red corpuscles remain in the center of the vessel, the surrounding space being occupied by clear plasma, in which an occasional leucocyte is seen, moving leisurely along the vascular wall. The minute capillaries contain few if any red corpuscles, and the stream in all the vessels moves along evenly and steadily, as attested by the behavior of the corpuscles. The first change in the vessels is that of contraction, probably a reflex action due to the stimulating influence of the local irritation. Soon, however, the vessels become dilated, and it is seen that the corpuscles are hurrying along with increased momentum. The red corpuscles increase in number rapidly, encroaching upon the space normally occupied by plasma and leucocytes. At first, the corpuscles hurry through the part as well as toward it, there being a largely increased amount of blood in motion toward, passing through, and moving away from the affected area (active hyperæmia). But after a time a slowing of the current becomes noticeable. The leucocytes now become prominent factors. Large numbers of them leave the axial current and join the slower procession along the walls of the small veins and capillaries, showing more and more of a tendency meanwhile to adhere and remain fixed to the inner surface of the vessel. Though the current may sweep them away again and again, they manifest a persistent tendency to return and adhere to the vascular wall. Finally, the entire inner wall of the small veins becomes paved with them,

Normal Circulation.

Inflammatory Engorgement of Blood-vessels.

and they seem piled upon one another in heaps, obstructing the lumen of the vessels in some places, causing complete arrest of the current (stasis). Careful inspection will now enable the observer to discover the fact that many of the leucocytes are passing, by amœboid movement, through the attenuated and damaged walls of the veins and capillaries. Some may be found just beginning the transit by sending a narrow prolongation through, while the bulk of the corpuscle is still within the vessel. Others may be found well on the way, a portion of each lying outside the vessel and a portion within it, the part engaged in the vascular wall being marked by a pronounced constriction, while others will be found wholly outside the vessels in the connective tissue.

To summarize, then, microscopical observation of a transparent membrane with *inflammatory engorgement*, will enable one to detect increased rapidity of the blood-current with subsequent retardation, dilatation and increased tortuosity of the minute vessels, migration of leucocytes (see illustration), diapedesis of red corpuscles, alteration of fixedtissue-cells, etc.

The changes in the circulation of the inflamed area vary in proportion to the position occupied in relation to the central point of irritation. At a considerable distance from the place of injury the circulation may still be normal. Nearer, the blood-vessels are dilated and the stream moves more slowly; still nearer is a zone in which there has been free emigration of leucocytes; and, when we reach the immediate neighborhood of the point of injury, the blood no longer flows through the vessels but remains stagnant, complete stasis having resulted. The meshes of the surrounding tissues are swollen, being distended by coagulable lymph, and the connective-tissue fibers are enlarged and softened. The epithelial cells of affected organs are swollen, their protoplasm is granular and more opaque, and fatty granules are frequently contained in them. Proliferation of new tissue gives rise to amœboid embryonal cells, which mingle with the leucocytes and red corpuscles that have exuded from the vessels. In many cases of inflammation, microbes play an important part, and are found in the leucocytes and red corpuscles, as well as in the plasma.

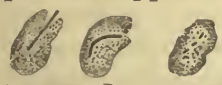
Cornea and Cartilage contain no blood-vessels, and, consequently, the vascular changes which are noted in other tissues are not observable, though in the case of inflammation of the cornea it will be noticed that vascular loops may spring from the sclerotic vessels and invade the plasma-channels, this being the result of inflammatory stimulation.

One of the first microscopical phenomena of *corneal* inflammation

is the appearance of migrating leucocytes in the plasma-channels, which are found in great numbers, packing the passages and moving toward the point of irritation. Simultaneously, the fluid contents of the spaces are increased in quantity, the spaces being distended and the cornea swollen, the tissue-cells becoming softened and opaque. The infiltration results from fluid supplied partly by adjacent blood-vessels, and partly from the tissue-cells of the affected structure. Vascular loops spring up from the sclerotic vessels at the periphery, and follow the leucocytes along the plasma-channels. Sometimes these become so prominent as to constitute what is known as *pannus*.

Cartilage possesses no circulating channels in its structure, its circulation being carried on by cellular diffusion, solely. Consequently, active inflammatory action does not occur, chondritis being a chronic process. The principal changes which occur early, therefore, can only be noticed as changes in the cartilage cells, which undergo enlargement, softening, and degeneration. After long-continued inflammation of cartilage, however, new vessels may grow over the affected surface, and even penetrate the substance of the cartilage and grow in the direction of the inflammatory focus, similarly to the vascular phenomenon presented in pannus.

Phagocytosis is a term applied by Metschnikoff to a destructive process supposed, by him, to be exerted upon microbes by leucocytes and certain fixed tissue-cells, such as mucous corpuscles, connective-tissue cells, endothelia of blood-vessels, alveolar epithelium of the lungs, and certain cells of bone, marrow, lymphatic glands, the spleen, etc. The process is accomplished by amœboid action, the cell folding or closing the microbe within its substance, and afterward destroying it by some process, probably digestive in character. Cells which are supposed to accomplish this action are termed *phagocytes*. Sometimes the microbes multiply in the cells in such numbers that the phagocytes are destroyed instead, and fatal results, general or local, follow. Against the testimony of Metschnikoff, who asserts that he has witnessed the operation of phagocytosis, we have the statements of other eminent pathologists that cells do not possess the power of destroying microbes which inhabit their substance. As there seem to be very positive statements upon both sides of this proposition, it is evident that the question requires future time for its complete solution.



Successful Phagocytosis.

Unsuccessful Phagocytosis.

Chronic inflammation is attended by much less vascular excitement than the acute form. Consequently, migration of leucocytes is lim-

ited, if occurring at all, and the inflammatory transudation is derived chiefly from the fixed tissue-cells, young cells here playing an active rôle. New tissue is developed, which, if it be not removed during the reparative stage, constitutes a permanent hyperplasia. Hyperplasia of connective tissue figures prominently in the morbid anatomy of interstitial inflammation of the liver, lungs, brain and spinal cord, choking out functional cellular structures in these organs, and strangulating, by slow process of contraction, the circulation of blood, upon which their functional activity depends.

Another result of chronic inflammation is the development of *granulation-tissue*, which is composed largely of embryonal cells, corresponding to the type of tissue in which or from which they grow, modified by disease-influences, such as the presence of microbes. The gummata of syphilis may be cited as an illustration of this class of growths.

TERMINATIONS OF INFLAMMATION.

The inflammatory process may be arrested at almost any step in its course, and the termination of the morbid action be consequently modified by the stage of arrest. If the irritation is not severe and the morbid action ceases before there has been destruction of tissue and purulency, the exudation is absorbed and the tissues are left in a normal condition, *resolution* having taken place. Long-continued inflammation, however, results in the death of the tissue-cells near the point of most concentrated excitement, and as there is liable to be a lodgment of many leucocytes here, we may have necrosis of tissue with purulent degeneration of the surrounding parts, constituting what is termed *suppuration*.

Pus consists of two parts: (1) Corpuscular elements derived from leucocytes and embryonal tissue-cells, which have undergone destructive changes; and (2) pus-serum, a fluid derived from the inflammatory transudation. The formation of pus depends, in infectious inflammation, upon the direct action of microörganisms upon the leucocytes and embryonal cells (unsuccessful phagocytosis), or upon the destructive action of the toxines generated by them upon these bodies. Pus, however, may arise from other causes, as certain chemicals injected into the tissues will cause similar changes. Severe inflammatory action from any cause may prove destructive to the elements which afterward degenerate into pus.

When purulent destruction occurs upon an open surface, and is gradual (and attended by molecular disintegration), the pus escapes freely, and constitutes *ulceration*. When a considerable portion of tissue is simultaneously involved in necrotic change, it is removed in a mass, and is termed a *slough*. If pus accumulates in the tissues, an *abscess* is the result. The disposal of the pus in such a case will

depend upon circumstances. If it be small in amount, and the surrounding tissues are in an excellent condition of health, the rather uncommon result of absorption may occur, the corpuscular elements being first liquefied. However, the pus is commonly evacuated by a burrowing process, the surface being reached through the least-resisting tissues, in a direction suggested by the force of gravity, in many instances.

Occasionally, pus may be inspissated, and retained in the tissues, the process being technically termed *caseation*. It suggests degenerative changes later on.

In chronic inflammation, the inflammatory action is not severe enough to destroy the tissue-cells, and they are stimulated sufficiently to undergo multiplication and rapid reproduction (proliferation). Connective tissue is especially prone to active proliferation of cells when chronically inflamed, and when the stroma of organs like the liver, kidneys, lungs, etc., becomes thus involved, the inherent contraction which follows the development of this tissue in the interior of an organ gives rise to obliteration of circulating vessels and parenchyma-cells, until the functions of the part may be completely destroyed, the organ becoming hardened, and presenting the condition known as *cirrhosis*.

The destruction of tissue following necrosis of cellular elements is usually replaced by proliferation of connective-tissue cells, which organize and fill up the vacancy. When this occurs upon the surface, a kind of epithelial covering—not exactly like the original one—covers in the new growth. New growths of connective tissue also fill up pus-cavities in the deeper structures, more or less contraction marking each point afterward The new growth is termed a *cicatrix*.

Symptoms.—In mild cases of acute inflammation, the local manifestations are the only observable symptoms; and these vary much, according to the part or tissue involved. However, in all acute inflammations there is a group of symptoms, more or less well-marked, not easily overlooked. These are, increased local heat, redness, swelling, pain, and impairment of function. In chronic inflammation, the disease may be so insidious in its progress that vital organs are fatally impaired before the patient is conscious that he is seriously ill.

In acute infectious inflammation, as well as in other cases involving important organs extensively, *constitutional disturbances*, which are usually well marked, accompany the local symptoms, and the local irritation is proportionally severe. The onset is liable to be marked by chilliness, if not by an actual rigor. Reaction is attended by pronounced febrile symptoms, such as elevation of the tempera-

ture of the general circulation, arrest of secretion and excretion to a certain extent, restlessness, etc. If the inflammatory action continues for several days with unmitigated severity, hectic fever and colliquative sweats begin to appear. A chill, occurring after inflammatory action has been established for several days, suggests commencing suppuration in some important organ.

One of the first prominent symptoms of inflammation is increased local *redness*, this being due to the active hyperæmia which occurs in the beginning. Though this may not always be a prominent feature, close inspection of vascular tissue will usually detect engorgement at the point where the active disturbance is progressing. Later, after the active stage has passed and the vessels become packed with corpuscles which move through the vessels but slowly if at all, the bright redness of the active stage gives way to a darker red, or purple hue.

The increased amount of blood in the part gives rise to dilatation of blood-vessels, general swelling, and augmented *local heat*. The elevation of local temperature is readily demonstrated when a thermometer is applied to the surface of the affected part and its temperature compared with that of other portions of the surface not affected. The local elevation has no direct reference to the general elevation, which is due to systemic infection and its influence upon the heat centers. The local elevation of temperature sustains a direct relation to the amount of blood in the part.

The inflammatory exudation is an element to be added to the local hyperæmia in the causation of the *tumefaction*. Fluids are poured out from the damaged vessels to distend the para-vascular tissues, and to these are to be added the transudation from the fixed tissue-cells, as well as the corpuscular elements which have escaped from the blood-vessels. Inflammatory transudation differs from the transudation of simple œdema, in that the inflammatory transudation contains albuminous elements, while these are absent from dropsical effusion.

Pain is a result of the swelling, which causes pressure upon the extremities of sensory nerves. However, the amount of pain is not proportionate to the amount of swelling, the resistance of the structure involved determining the amount of pressure and consequent compression of nervous structure. Loose tissues may be swollen remarkably and yet not be very painful, while inflammation of firmer parts may give rise to excruciating pain, and not present much of a swollen appearance. The *resistance* offered to the inflammatory exudation determines the amount of pain, to a great extent. The pain of acute inflammation is usually throbbing in character, this being due to increased pulsation of all the minute arteries, as well as to

the exalted sensibility of the nerves of the affected part. Accompanying the throbbing sensation may be paroxysms of darting or burning pain.

All the symptoms of inflammation may be fully developed within twenty-four hours after commencement, though usually a longer time is consumed in its full development, and the symptoms may be progressive for several days, in severe infectious cases. The extent to which a part may become involved will depend upon the virulence of the existing cause, partly, and partly upon the receptivity of the tissues to its action.

Tenderness on pressure is a very important symptom of inflammatory action. Sensitiveness is a condition which is almost always present, even if the part be painless when undisturbed. In some cases the pain is reflex, the irritation being manifested in a part distant from the real point of morbid action. In morbus coxarius, for example, the inflammation is in the hip joint, while the pain is in the knee. Pressure upon the trochanter in such a direction as to crowd the head of the femur into the acetabulum will elicit tenderness, while no ordinary amount of pressure about the knee will cause discomfort. In some cases of proctitis, the pain will be in the hip, along the sciatic nerve or in some other remote part, while pressure about these regions will fail to elicit tenderness, and the actual seat of the disease is only demonstrable after careful examination of the rectum. The pain of endometritis may be persistently manifested in the ovarian region, but tenderness will not be discovered until the uterine cervix is disturbed.

In some cases, impairment or perversion of function may be the only prominent symptom of inflammatory action. The almost complete arrest of the urinary discharge in acute Bright's disease may be the first noticeable symptom. The *gravity* of a case may not depend so much upon the comparative amount of tissue destroyed, as upon the character of the function impaired. For example, the tissue-destruction involved in a fatal case of pneumonia might not be of such serious consequence were it not for the arrest of the important oxygenating functions of the parts involved. And arrest of this function will give rise to the leading symptoms of the case, such, for instance, as hurried respiration, dyspnœa, cyanosis, cough, expectoration, etc.

It thus becomes apparent that any attempt to describe the diversified symptoms of inflammation within the limited scope of a single article, must be rambling and unsatisfactory. Indeed, much of the space in the following pages will be occupied in the consideration of the symptoms of various inflammatory conditions.

Treatment.—The diversified conditions liable to be met in a variety of cases of inflammation, render it inexpedient to attempt to cover the ground occupied by the proper treatment of individual forms in this place. This will be the task to be fulfilled in the pages which follow. However, there are certain well-established principles to be observed in all instances, and a consideration of these will assist the practitioner very much in individualizing his cases.

There is much to be considered in a proper *regiminal treatment*, in discussing the general management of inflammation. Provisions against irritation of an already inflamed part are as important as curative means, and these often amount to as much if not more than medicinal treatment. Rest to an inflamed part means much when its activity augments inflammatory action, as is often the case.

Rest being important in treating inflammation of any part, the questions arise, What does it constitute, and how shall it be attained? These the physician of practical turn will nearly always be able to solve by the application of common-sense principles to individual cases. It is axiomatic that neither excessive functional activity nor abnormal irritation of an organ or part should be allowed to continue longer than salutary measures will suffice to repress it.

To illustrate, imagine a case of recto-colitis, in which the evacuations are frequent, and attended by severe tormina and tenesmus. Suppose now that the patient be allowed to rise and sit upon a stool at every period of evacuation, thereby adding to the irritation by change of position and by voluntary straining. In such a case—which is no uncommon illustration of the management pursued by many—the therapeutist may find his best prescriptions at fault many times, and will occasionally find his patient growing worse instead of better, until he has enjoined quiet in the recumbent posture with the use of a bed-pan during evacuation, and instructed the patient to exercise the will-power to postpone the attempts at evacuation as long as possible, in order that straining and other causes of hyperæmia thus entailed may occur only at prolonged intervals. Prompt response to the properly selected remedies will then follow, and the benefit of rest to the affected part will become so prominent that no one can doubt its presence. Take, again, a case of irritation of the respiratory mucous membrane arising from measles. Suppose, now, the patient be allowed to remain during the course of the disease in an apartment where the temperature is below the freezing point, the cold air acting as a local excitant to the irritated surface. The best remedies we may select here cannot equal, in beneficial results, the adoption of means to bring the temperature up to 65° or 70° F. and maintain it there, during the continuance of the bron-

chial irritation. Indeed, neglect of this measure may result fatally in cases which would terminate favorably under proper surroundings without any medication at all, pulmonary inflammation being excited by the irritation set up from the excessive cough, arising from exposure of the pulmonary membrane to the chilly atmosphere. Suppose a case of chronic laryngitis, due to the titillating influence of an elongated uvula. Could it be reasonably expected that medicine would cure the disease while the cause of irritation was remaining?—Certainly not. And the physician who possessed such an exalted opinion of remedies as to expect it, would be blind to the true philosophy of therapeutics.

The rest which irritated and inflamed organs receive from *opiates* is seductive, and usually of little permanent good, while the effect of the drug is often harmful to the general condition of the patient, impairing his recuperative energies. However, this favorite method, long perpetuated by the dominant school, is not to be abandoned completely, though its omission should be the rule rather than the exception.

Position may exert an important influence upon the results of inflammatory action, when this operates upon the circulation of the affected part. Hypostatic pressure is influenced by gravity, even within the body; and flexure of certain parts may compress important blood-vessels to impede the circulation, when the force of gravity is not at fault. Destructive and fatal pneumonia may arise during typhoid fever, from allowing the patient to remain constantly upon the back for weeks at a time, the fatal effects of hypostatic pressure upon debilitated tissues being here demonstrated. Chronic metritis may be due to flexion of the uterine cervix impeding the circulation of blood through the uterine vessels.

Undoubtedly, inflammatory conditions of the intestinal walls are often aggravatad by the local effect of *improper food.* Fatal cases of typhoid fever may owe their unfortunate termination to such influences; and dysentery, which, under proper management, would terminate favorably in brief time, may be prolonged until permanent chronic disease is the result, because proper attention has not been given to the fact that a local hyperæmia exists in the intestinal mucous membrane, which renders careless and indiscriminate feeding highly improper and detrimental.

These few illustrations will suggest the proper course to the discriminating practitioner. Give therapeutics their proper place, and do not expect them to accomplish impossibilities.

It is evident, then, that many more details are to be considered in the management of inflammation generally, than those which concern

the administration of remedies; for, though these are highly important, neglect of a proper regimen may neutralize the best-directed therapeutic efforts, and subject the most reliable remedies to condemnation.

In the *therapeutic* management of inflammation, we must be guided by the stage which has been reached in pathological development, the relief of irritation and conservation of the vitality of the part affected being the principal objects sought. We will leave the destruction of pathological microbes concerned to the bacteriological enthusiast and neophyte, and concern ourselves, in this direction, with neutralizing the ptomaïnes generated, and reënforcing physiological processes, so far as possible.

Let us first consider the treatment of *active hyperæmia;* for, in the proportion that this can be controlled, in a corresponding ratio will the integrity of the tissues involved be preserved. Two classes of remedies are to be considered here, namely, (1) *general vascular sedatives*, and (2) *local vascular sedatives.*

The **general vascular sedatives** have already been considered, under treatment of fevers. They include the special-sedative class of Scudder, and the antiseptic sedatives. These are to be employed to control the symptomatic fever, usually, though they should not be considered mere appendices of this character, as inflammations in which constitutional symptoms are not prominent are manifestly modified by them. Indeed, the hyperæmic conditions of inflammation may be successfully treated without other means, except properly selected local applications. As the appropriate method of employing these remedies, as well as their adaptation, has already been discussed, the reader is referred to preceding pages (10, 11, 12, 13, 14), and to Dynamical Therapeutics, for further suggestions.

However, we can improve upon this treatment by adding a class of remedies—**local vascular sedatives**—which sedate special localized vascular areas. For example, while we may treat pneumonia with tolerable satisfaction by the use of aconite, gelsemium, jaborandi, and other general vascular sedatives, we will derive better satisfaction by combining with the properly selected general sedative, a remedy from the group which sedates the vascular area supplied with blood by the bronchial arteries; such, for example, as asclepias, bryonia, ipecac, lobelia (?), etc. So with acute pharyngitis. The general sedatives may control the local vascular excitement, and the results be very satisfactory to those accustomed to older and less direct methods; but more rapid and satisfactory results follow when phytolacca, cistus canadensis, or some other agent which specifically influences the vascular area supplying the

pharynx is added for its sedative effect. The local sedative usually acts with greater therapeutic power upon the special part than the general sedative, but the *combination* acts best to cover both general and local disturbances of active hyperæmia. If we make a careful study of dynamical therapeutics, we will find that many different parts and organs possess their specific vascular sedatives, which exert an important influence in controlling hyperæmic conditions.

By the use of such agents we may be enabled to so control the active hyperæmia of the affected part as to quiet the inflammatory action before the later stage, such as migration of leucocytes, inflammatory engorgement, stasis, alteration of fixed tissue-cells, etc., has made much progress. Consequently, there is little exudation to be absorbed, and the blood-vessels and fixed tissue-cells are saved from damage, while necrotic processes are averted, recovery then being rapid and complete. It matters little how severe an inflammation may be, faithful adherence to such principles is sure to provide against the worst results, which might ensue without their observance.

When the para-vascular tissues become involved, the disease has progressed beyond the reach of vascular therapeutics. However, as para-vascular disturbances may go on simultaneously with hyperæmia, it is not in order to abandon our sedatives upon the adoption of a new line of treatment. These should be continued, and alternated with additional measures, as long as active hyperæmia persists.

The period of commencing inflammatory exudation announces the time for a new step in the treatment of all severe cases of inflammation. Wherever the formation of pus would be disastrous, as in inflammation of any internal organ,—where its exit might be attended by serious consequences,—it is highly important that this purulent degeneration be forestalled and prevented, if possible, as it often is. In many cases of pelvic cellulitis, typhlitis, hepatitis, pneumonia, etc., where purulent accumulation might otherwise occur, **potassium chloride**, 3x trituration, properly employed, will assist the normal processes to remove and dispose of inflammatory exudation so safely as to leave no bad results behind, and convert portentous cases into that favorable form where resolution disposes of serious sequlæ. It is remarkable how soon pain, hectic fever, and tenderness will vanish before this remedy, in a large majority of cases. True, weeks may be required to bring about the desired results in some cases, and the remedy may fail, as all others will; but it is one of the most precious boons of modern therapeutics, after all. Add five grains of the 3x trituration to half a glass of water, and give a teaspoonful every hour.

A step further, and we find that purulency cannot be controlled. We now need a remedy which will hasten the change rapidly, that as small a portion of tissue as possible may be sacrificed; and we will resort to *calcium sulphide*, for this purpose. When there is a tendency to persistency of suppurative action, this remedy will often assist promptly in bringing the degenerative change to an end, as, for instance, where there has been purulent pneumonia, and the pus cavity continues to suppurate after evacuation, preventing the maturity of embryonal tissue-cells, and the repair of the part.

Then we have more extreme cases, where a sloughing tendency is announced by purple tissues, with darkened center, or perhaps actual necrosis of the focus of inflammatory action, with manifest progression of the necrotic change, where none of the remedies already named will be of much if any account. It is here that we will expect to derive the wonderful influence of **echinacea**, employing it both internally and locally, saturating the system with it, and stimulating local areas by its direct action. Nothing like this remedy was ever known in medicine before its time, and the physician who neglects to avail himself of it is sacrificing the vital interests of his patient where marked necrotic tendencies are developing. From ten to twenty drops of a saturated tincture of the fresh plant may be given every hour, and a dilution of one part of the same to three or four of water should be applied locally at frequent intervals. Another remedy of this character is *baptisia*, a traditional agent for such conditions, and one which deserves much praise, though it does not compare with echinacea in efficacy.

Then we have a class of inflammations where the skin and its reflections are involved in erythematous or erysipelatous irritation, the condition being marked by tendency to rapid spreading, and severe burning pain. Here we get the best effects from *echinacea* internally, though it may be assisted by *aconite*, *jaborandi*, or some other appropriate special sedative, and its local influence, which is most effective, may be assisted by applications of *plumbi acetatis*, *citric acid*, etc., in solution. Where such conditions become chronic, the grand constitutional influence of *berberis aquifolium*, should not be forgotten.

The *symptomatic fever* which attends inflammation will call for the medication already directed under the general treatment of fevers. The sedatives, both arterial and antiseptic, should be properly adapted when called for; and the element *periodicity* should be recognized and properly met, if success is to be expected to follow treatment.

The *local* treatment of inflammation has undergone quite a revolution since the days of bacteriology. Hot poultices, once the favorite resort of the practitioner, have been relegated to the past, their

use being opposed to antiseptic precautions, it being believed that they furnish a nidus for the development of pathogenic germs. *Cold*, applied over the affected surface, is less conducive to suppuration, and water may be used to saturate appropriate packs for this purpose. The temperature of the water employed should depend upon the patient, one of delicate nervous organization not being well adapted to resist the shock of very cold applications. Tepid water would suit such individuals best, and impart all the beneficial influence to be derived. In pneumonia, especially in children, cold- or tepid-water packs should rank among the best means of treatment during the stage of active hyperæmia; and such remark applies to acute inflammatory action in almost any other organ. Where superficial surfaces are involved, some appropriate medicine may be added to the water, which will serve both as a therapeutic agent and vehicle to carry the medicine. For instance, superficial inflammation of the skin may require the local influence of diluted carbolic acid, echinacea, citric acid, or some other cooling, soothing, or cleansing agent.

The *diet* of inflammation should be nutritious, but not stimulating. The old idea that inflammation should be starved out, was a much mistaken one, and has long since given way to more sensible views. The waste of tissue and expenditure of heat involved calls for nourishment to make good the loss, and demands that the patient be properly fed.

In active inflammatory states of severe character, a diet of milk may be all that is desirable, this being diluted one-half with water or Vichy. A better diet here will be found to consist of malted milk, though the patient soon tires of this form of food. From six to seven ounces of milk may be administered every two hours. Where the alimentary canal is involved, the diet must be selected with reference to existing conditions, care being observed to avoid everything liable, from its indigestibility or mechanical influence, to irritate the sensitive mucous membrane. Malted milk or beef-peptonoids will here be found excellent, preference being given to malted milk. Gruels, prepared from arrowroot or oatmeal, sometimes break the monotony of a continuous milk diet, and serve an excellent purpose as nutritives. Ricewater is very nourishing, and serves at the same time as a cooling drink in inflammatory diseases. The cream from cream-codfish is excellent, and unobjectionable during the later stages, after inflammation has somewhat subsided. At this time the yolks of eggs which have been boiled an hour, will be found nourishing and unirritating. Eggnog—though alcoholic stimulants are rarely demanded—may now be administered sparingly.

When acids are indicated by the tongue and craved by the patient, they are excellent in the form of acidulated drinks, such as lemonade, acid phosphate, etc. Later on, a light diet of toast, poached eggs on toast, custard, rice, etc., may be indulged in. Tea and coffee should be used sparingly, if at all.

HYPERTROPHY.

HYPERTROPHY is enlargement of an organ or part from increase in the size or number of its numerical elements. Simple increase in bulk, however, may occur without constituting hypertrophy, as proliferation of cells of new growth or of connective tissue may occur to increase bulk, without adding to the function-elements. Various degenerations may also increase the size of a part, which could not then be considered as hypertrophied.

In order that a muscle may be hypertrophied, there must be an increase in the size or in the number of the muscle-cells. In hypertrophy of the thyroid gland, there must be an increase in the number or size of the normal cellular elements of the part; therefore, some cases of goitre are true hypertrophies, while others are due to degeneration or hyperplasia. In true hypertrophy the enlargement must be due to increase in the normal cells of the part.

Normal hypertrophy frequently occurs, as, for example, when there is an increase in the elements of the uterine structure and of the mammary glands during the developments of gestation. The hypertrophy of the muscles of the calf in the ballet-dancer, of those of the forearm in the blacksmith, etc., is of a similar nature, though in every instance it must be considered as *compensatory*—a development in keeping with the requirements of the case. An example of compensatory hypertrophy is afforded by the hypertrophy of the heart when obstruction of the orifices demands the exhibition of greater power to propel the blood through them in a given space of time.

Irritation, which invites undue afflux of blood to a part, may result in hypertrophy. The enlargement of the cheeks and nose in acne rosacea probably depends upon this principle. In inflammation, the surrounding area is stimulated to greater than normal activity, and hyper-growth of normal tissue may result, as enlargement of bone in periostitis. The process by which hypertrophy develops from the normal elements of a part is one of cell growth, of which there are two kinds, namely, direct and indirect. Indirect cell development is technically termed *karyokinesis*. Recent advances in biology have improved our knowledge of the minute structure and developmental history of cell growth in this particular.

Modern knowledge of cell structure differs materially from the conception entertained by older writers, such as Schwann, Remak, and Virchow, who held that a structureless mass of protoplasm containing a homogeneous nucleus was the essential feature. Some writers even contended that a nucleus was not necessaary, a simple mass of protoplasm representing, in certain instances, an individual cell. For the purpose of karyokinesis, however, a much more complicated structure is essential, and more complete researches into the minute structure of the histological formation of cells have demonstrated that a nucleus with internal organization is an important essential of this process.

Successive steps in Karyokinesis.

The nucleus contains a reticulum of minute fibers, the meshes of which are filled with a homogeneous substance. From the fact that the fibers may be stained with certain coloring matters they have been termed *chromatin threads*, while the homogeneous substance it contains, resisting colors, is termed *achromatin*. The cell contents outside the nucleus also contain fibers, irregularly distributed through its substance. When the cell is in a quiescent condition the chromatin threads are very slight; but when karyokinesis begins, they become swollen, and converted into a skein of convoluted fibers. This afterward assumes the shape of a star, the wall of the nucleus meanwhile disappearing. Then follows the equatorial stage, in which the chromatin fibers divide into two groups and cluster about the poles of the nucleus, a clear space being left along the equatorial line. Then the cell wall contracts in this region and a separation of the two parts follows, the chromatin threads in each cell subsiding into the former condition of quiescence, and becoming surrounded by a limiting membrane.

ATROPHY.

Atrophy is a diminution in the size of an organ or part, due to loss of substance in its histological elements, or decrease in the number of such parts.

Where the decrease is only that of size of elemental parts, the condition is termed *simple atrophy;* where there is loss in the number

of such elements, it is termed *numerical atrophy.* As numerical atrophy must be the result of previous diminution in size in the elements which have disappeared, it is evident that simple atrophy must precede and accompany numerical atrophy, the two often being associated, though simple atrophy may occur alone. It must be patent that numerical atrophy is of the more serious character, as, when the histological elements of a part are destroyed the condition will be permanent, unless new cells are created—something not likely to occur—while in simple atrophy, under favorable circumstances, the elements may be restored to their former condition.

A familiar example of *simple atrophy* is loss of the subcutaneous adipose tissue which attends general emaciation. The adipose tissue here consists of connective-tissue cells filled with fat. When the fat is absorbed, the cells diminish in size, the general bulk of the body thus becoming wasted. In a similar manner the fat may be removed from the connective-tissue throughout the body, and portions of the contents of cells of other structures, thus resulting in diminution of bulk. The cells of glandular organs may thus be involved, such parts as the liver, kidneys, mammary glands, spleen, testicles, lymphatic glands, and other organs becoming wasted in size in this manner. The primitive fasciculi of muscles may also be thus affected, this being common in the heart and voluntary muscles, during wasting diseases. When restitution occurs, there must be an increase in the nutritive activity of these parts, and supply of more nutritive material.

In *numerical atrophy,* the loss of substance and lack of nutritive supply results in molecular disintegration of the elementary cells, this usually occurring in circumscribed areas, and only a granular débris finally remains, to mark the focus of atrophic action. This differs from necrosis, in that the substance is absorbed and carried away gradually in atrophy, while in necrosis there is such rapid death of the part that its substance remains as a foreign body, subject to immediate expulsion, the granular débris of atrophy still remaining a part of the living tissue, though its bulk be lessened and its function destroyed.

Atrophy may be *general* or *partial.* In general atrophy, all the organs and tissues, to a greater or less extent, are involved in loss of substance, while partial atrophy is limited to separate parts. General atrophy is usually simple, only the size of histological elements being involved, while partial atrophy is often numerical, certain histological elements being completely destroyed.

A better conception of the different forms and conditions of atrophy may be had by considering the *causes* of general and partial

atrophy. These may be summed up under three general heads: (1) *Deficient supply of nutritive material;* (2) *excessive waste;* and (3) *impaired nutritive activity.*

Any condition of affairs which interferes with the supply of sufficient nourishment is soon followed by wasting of the entire body. *Starvation,* insufficent food supply, soon manifests itself by emaciation and general atrophy of all the tissues of the body, the adipose tissues first wasting, the firmer structures being involved later. Conditions of the system which operate to interfere with the *appropriation* of food bring about the same result, as, for instance, obstruction to the passage of food into the stomach or intestines, such as stricture, or interference with the absorption of the food after digestion, as disease of the mesenteric glands, liver, etc. Any condition which prevents proper nutritive pabulum from reaching the blood in a digested condition will cause general atrophy.

Excessive waste of normal tissue-elements also results in general atrophy. We observe this in continuous hemorrhages, in diabetes, albuminuria, prolonged and profuse suppuration, and in the destruction of tissue which attends febrile disease.

Impaired nutritive activity is the usual cause of the atrophy of old age—senile atrophy. As age advances the plastic power of the tissues diminishes; and they are unable to appropriate nutritive material vigorously; and there is also enfeebled circulation. The result is a general wasting of the tissues, slow but permanent.

These causes are usually combined in the bringing about of emaciation. For example, in the marasmus of phthisis there is excessive waste from the colliquative sweats and profuse expectoration, the digestive organs are so involved that little food is consumed, while the assimilative powers are impaired. And so with almost every other condition of general atrophy except starvation from lack of food. Disturbance of one organ or function begets disturbances of others, and a chain of circumstances results from the combination, all of which tend toward loss of tissue—general atrophy.

The *causes* of *partial atrophy* are: (1) *Imperfect supply of blood;* (2) *diminished functional activity;* (3) *increased functional activity;* (4) *the action of certain drugs;* (5) *nervous influence;* and (6) *inflammation.*

Imperfect supply of blood is usually the result of pressure upon the arteries concerned in carrying nutritive material and distributing it to the affected part. This may arise at a distance from the atrophied organ or area, from pressure to a main trunk from the growth of a tumor or contraction of cicatricial tissue, or it may be due to the proliferation of connective-tissue elements of the stroma of an

organ with subsequent contraction, resulting in strangulation of the normal circulation. Direct *pressure* upon a part which is not yielding may result similarly, as when an aneurism, or even an arterial trunk, presses against the surface of a bone, causing indentation and atrophy of its tissue in that particular place. The pressure of hydrocephalus within the cranium causes thinning of the cranial bones; that of retained secretion in the bladder from urethral obstruction, atrophy of the kidney, etc.

Diminished functional activity is a common cause of atrophy. Disuse of the muscles of locomotion is soon followed by atrophy of these parts. Let a person remain in bed for a few weeks, even when in fair bodily health, and the muscles of the lower extremities become very much lessened in size. The disuse of fœtal organs which follows the changed conditions of birth results in rapid and complete destruction of the functional capacity of the ductus arteriosus, the umbilical arteries and veins, and the Wolffian bodies. The involution of the uterus after parturition, and the wasting of the lower jaw after loss of the teeth, are other examples of physiological atrophy from disuse.

Groups of paralyzed muscles soon atrophy, though in some cases this may not be due to loss of function alone, trophic influences being brought more or less to bear; for there are evidently certain nervous filaments which connect nutritional centers in the spinal cord with every part of the body.

After establishment of an artificial anus, the rectum becomes atrophied, often dwindling away to a mere fibro-cellular cord. Section of nerve trunks is followed by atrophy of the distal extremity, and atrophy of the optic nerve follows enucleation of the eye or destruction of its function-elements.

Increased functional activity is occasionally, though rarely, a cause of atrophy. Some glandular structures, especially that of the testicle, dwindles away as a result of excessive activity.

Certain drugs cause atrophy of particular organs. Iodine causes more or less wasting of the lymphatic glandular system; bromine, of the testicles; and fucus vesiculosus and the juice of phytolacca berries are said to produce atrophy of adipose tissue.

The atrophy which results from *inflammation* is usually due to the organization of new fibrous tissue in the stroma of organs, the contraction of which compresses the circulation and thus cuts off or impedes blood supply to such an extent as to deprive the affected parts of normal nourishment. Dwindling away of the elements of an affected organ therefore results, and, when this is a vital organ, like the liver or kidney, fatal results follow.

There is often the association of fatty degeneration with atrophy, the same condition—interference with normal supply of nutrition—operating to bring about both these results at once. During senility both conditions are often associated, atrophy and fatty degeneration occurring together as results of limited blood supply to the affected part. Brown atrophy of the heart is an instance of the association of these conditions. It consists of gradual atrophy of the muscular fibers, attended by the formation of brownish yellow or blackish pigment. The fibers are often at the same time the seat of fatty degeneration. Association of atrophy and fatty degeneration are not uncommon in pulmonary tuberculosis, pernicious anæmia, and other wasting diseases.

It is not probable that all cases of atrophy may be benefited by treatment; indeed, comparatively few of them can be improved. Correction of the conditions which lead to them is the first thing to be thought of, though this is not always possible. In some cases the condition may be modified, at least, by the judicious use of electricity and massage, these measures encouraging the circulatory and nutritional activities. The action of certain drugs improves the plastic power of atrophied parts, though the subject requires a further investigation. For example, sabal serrulata influences the mamary glands and testes in this manner, increasing their size and functional power. Bryonia exerts a similar influence upon the retina and optic nerve. Collinsonia thus influences the rectum, and rhamnus californica the muscles.

III. DEGENERATIONS.

The degenerations differ from atrophy, in that there is alteration in the *quality* of the cells of an affected part which not only impairs but destroys their functional capacity. Complete annihilation of a part may thus result, its character being histologically as well as functionally altered.

Two kinds of degeneration occur, which are described as (1) *metamorphoses*, and (2) *infiltrations*.

The *metamorphoses* are the result of a direct change in the albuminoid constituents of the cells of a part, by which they are converted into a new material. This is attended by a complete destruction of the intercellular substance, which softens and loses its normal characteristics, the entire normal structure of the part being annihilated.

The *infiltrations* are characterized by the infiltration of new elements into the cells of a part, which displace the normal elements to

a certain extent, but which does not destroy them nor interfere completely with their functions. The intercellular substance is not usually destroyed, and the affected part may retain a modified portion of its structural and functional individuality.

Parenchymatous Degeneration.

The common name for this form of degeneration is "cloudy swelling," though it is otherwise known as albuminous, serous, and granular degeneration. It consists of a swelling of the anatomical elements of portions of the body, accompanied by granulation of the cell-contents, and disappearance of the nuclei. The granules resemble fat-granules in appearance, but differ from them by being soluble in acetic acid, and insoluble in alcohol or ether. The parenchyma-cells of important organs, such as the liver, kidneys, heart, etc., are especially prone to such a condition, the organs becoming swollen, pale, and friable. Cells which undergo this degeneration are not necessarily destroyed, a gradual return of their integrity following favorable constitutional conditions.

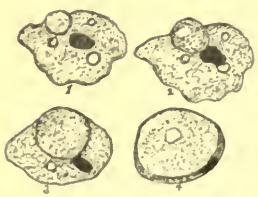
Cloudy Swelling.

This form of degeneration attends many severe acute diseases, the infectious fevers being especially prone to it. All soft tissues participate, though the abdominal and thoracic organs and kidneys suffer most. It may follow poisoning from arsenic, phosphorus, or the mineral acids.

Cloudy swelling occurs especially in epithelial elements. The accompanying illustration represents progressive stages of degeneration of this character in the epithelial cells of the urinary tubules, in acute tubal nephritis.

Fatty Infiltration.

Fatty infiltration consists of infiltration of fat into cells in such a manner as to displace the nucleus and crowd aside other elements without destroying their functions. The fat accumulates within the affected cells as distinct globules, displacing the nucleus and protoplasm, though after it is removed by absorption the cell remains in a normal condition. Fatty infiltration occurs as a normal process, due to the presence of more fatty material in the body than is required for physiological purposes, the excess then being stored in the connective tissue for future use. The favorite



Fatty Infiltration.

points for fatty infiltration are adjacent to the radicles of the nutrient vessels.

In fatty infiltration the fat is derived from the oleaginous, saccharine, and nitrogenous principles of the food, instead of from the affected tissues themselves, as in fatty metamorphosis.

Abnormal fatty infiltration occurs in obesity and emaciation. In the one instance, there is more fat in the body than the natural oxidizing powers are capable of destroying; and, in the other, the oxidizing processes are so interfered with that the fat, in even a small amount of food, is incompletely oxidized, accumulation of fat in the cells resulting. Thus, in chronic phthisis, we may find that fatty infiltration is often present.

The accompanying illustration represents the gradual infiltration of a cell with fat, with displacement of the normal cell-contents.

Fatty Degeneration.

Fatty degeneration, or fatty metamorphosis, differs from fatty infiltration in that there is an alteration of the normal cell-contents instead of infiltration of fat, the normal cell-elements thus being destroyed, the albuminous constituents of the tissues themselves being converted into fat.

Fatty Degeneration of the Heart.

Thus the cell furnishes the fat for its own substance, minute granules or globules of fat making their appearance in the cell, the entire protoplasm finally becoming converted into fat-granules.

States which interfere with the proper quality of the blood, so that the tissues are imperfectly nourished, as in pulmonary tuberculosis, protracted anæmia, and other wasting diseases, tend to general fatty degeneration, certain tissues, such as the heart, liver, kidneys, walls of arteries, and voluntary muscles, being especially prone to fatty deposits.

The metamorphosis of fatty degeneration begins in the protoplasm of the cells, outside the nucleus—though this is soon involved and broken up—as transformation of minute particles of the albuminoid substance into fat-granules. These multiply, and the destructive change invades the nucleus, transforming its substance into fatty granules and obliterating its limiting membrane. As fatty granulation progresses in the cell, its entire contents become fatty, the change involving the cell wall and intercellular substance. Neighboring cells becoming involved and the intercellular structure becoming disorganized, the fatty remains of numerous cells may coalesce to form fatty masses, which exist at the expense of the normal tissue invaded.

The accompanying cut represents a section of the cardiac muscle which has undergone fatty degeneration. The striæ are seen broken up in places, their substance having been converted into fatty granules, which are distributed more or less profusely throughout the structure.

Parts which are insufficiently supplied with blood on account of pressure upon nutrient arteries are prone to this form of degeneration, the coronary arteries being partially occluded by atheromatous products when fatty degeneration of the cardiac muscle occurs.

Separation of nerves results in fatty degeneration of the distal extremities. Organs which have become atrophied from disuse are prone to fatty degeneration, as a result of diminished blood-supply and imperfect oxidation. The tissues of old persons are liable to such changes, due to impoverishment of blood-supply, the metamorphosis occurring in the cartilages, cornea (arcus senilis) and lens (cataract), and in the brain (cerebral softening).

Mucoid and Colloid Degeneration.

These forms of degeneration resemble each other so much that they are very liable to be confounded, though there is a material difference in the nature of the two. Mucoid degeneration is more liable to affect the intercellular substance, while colloid degeneration is more liable to affect the cellular elements. Colloid material differs from mucin, the product of mucoid degeneration, chemically, in containing sulphur, and in not being precipitated by acetic acid.

Mucoid Degeneration. This process has its normal type in the secretion of mucus by the mucous follicles, the epithelial cells here being converted into mucin and cast off, forming a transparent gelatinous substance, familiar to every observer. Mucoid tissue constitutes the earliest form of every fœtal structure, a higher development being taken on later, though the mucoid material persists in the vitreous humor of the eye throughout life, and is familiar to the obstetrician in the structure of the umbilical cord.

Other structures than the mucous membrane may develop this substance (mucin) under abnormal conditions. Connective tissue, cartilage, bone, marrow, adipose tissue, and sarcoma (a new growth), may undergo abnormal mucoid softening. This is liable to occur simultaneously in considerable patches, the basement membrane becoming involved, the fibrous structure contained in it being converted into homogeneous material. The tissue-cells may then persist, undergo fatty degeneration, or partake of the mucoid change.

Mucoid degeneration most frequently occurs in cartilage, especially the intervertebral and costal cartilages of old people. Con-

siderable cavities of cyst-like accumulations of mucin may be found here as the result of such change. The myxomata or mucoid tumors consist of this material. The causes of this form of degeneration are unknown.

Colloid Degeneration. This differs from mucoid degeneration in the fact that the cells are the parts especially involved. The albuminoid material in the cells becomes converted into colloid, this first appearing as minute lumps in the interior of the cell, which gradually increase in size, crowding the nucleus aside, and finally representing the entire cell structure. This constitutes a colloid mass, which swells up and bursts the cell wall, to coalesce with contents of neighboring cells which have undergone a similar change. This coalescence results in the formation of cysts or accumulations of greater or less size, of a gelatinous, shining, transparent material, resembling cooked sago in appearance, though of a yellowish color and tolerably firm consistence.

Colloid changes occur most frequently in enlargement of the thyroid and lymphatic glands, in the choroid plexus, and especially in the new growths.

Amyloid Degeneration.

This term, with our present knowledge, is evidently a misnomer, though when Virchow applied it he supposed, from the resemblance of the chemical reaction between this substance and iodine to that between iodine and starch and cellulose and iodine, that it belonged chemically to the starchy group, and named it "amyloid" (like starch). But later investigators have demonstrated that the substance of amyloid degeneration is a nitrogenous formation, an albuminoid, and not in any way related to starch.

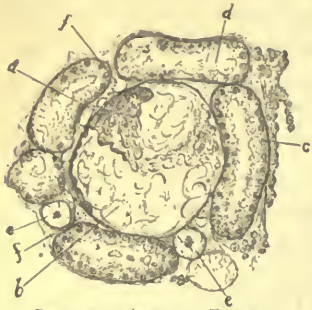
Its reaction with certain matters, however, is remarkable. When a solution of iodine is added to it, it becomes colored mahogany brown, the surrounding unaffected tissue appearing pale yellow. Iodine and sulphuric acid impart a blue color to it, and methyl-blue and gentian a bright red or pink color.

To the naked eye, the morbid deposit appears semi-translucent, waxy, or lardaceous—resembling the fatty portion of fried bacon. When it invades internal organs extensively, they become swollen, resistant to pressure, increased in weight, their capsules being tense, dry, and pale. When it invades the spleen, the cut or broken surface resembles boiled sago, and the condition has been commonly termed "sago spleen." The liver, kidneys, lymphatic glands, serous membranes, mucous membrane of the alimentary canal, blood-vessels, and connective tissue, are all liable to the invasion of this degeneration.

It accompanies depraved states of the blood due to prolonged

disease, such as tuberculosis, leukæmia, suppuration of bone, chronic dysentery, etc., though it is absent in cancer. It occasionally follows inflammatory action. It is very liable to develop during senility, without other apparent provoking cause.

The character of the deposit is not yet well determined. The consensus of opinion seems to be in favor of the belief that the deposit is fibrin which becomes separated from the blood by metamorphosis or infiltration. Color is added to this theory by the fact that the disease nearly always begins about the capillaries and minute arteries, the deposit appearing in the peri-endothelial fibrous tissue coating the outer side of the endothelial wall. If this occur in internal organs in which the parenchyma-cells lie within capillary plexuses, the organ-cells may remain unaffected while the capillaries are extensively involved. If this occur in the liver, the liver cells may remain unchanged between the masses of amyloid material which are deposited about the capillary plexuses surrounding them. So with the kidney; the amyloid deposit is massed about the capillaries of the glomeruli and other minute vessels, while other histological elements remain unchanged; though in some instances the connective tissue of a part, such as the spleen and lymphatic glands, may suffer most.



Section of Amyloid Kidney.
a, Norma capillary loop.
b, Amyloid capillary loop.
c, Normal epithelium.
d, Hyaline tube cast.
e, Amyloid arteriole.
f, Amyloid capillary.
g, Loosened fatty epithelium.

Fatty changes are liable to attend amyloid degeneration, this being due to obstruction and compression of the blood-vessels.

Certain concretions called amyloid bodies appearing normally in the brain, prostate gland, and vesiculæ seminales, afford the same color reactions with iodine, and iodine and sulphuric acid. They bear no relation to this form of degeneration, and are probably a normal accompaniment of advancing years.

Calcareous Degeneration.

This is an infiltration, not a metamorphosis. It consists of the infiltration of normally soft tissues with calcareous and magnesian salts, rendering them brittle, chalky, and to the touch, gritty. It occurs as a normal process when calcareous elements are deposited in cartilage preparatory to the formation of bone, but here the infiltration is succeeded by organization of the calcareous material into cells and bone tissue, while in degeneration, the calcareous material remains as an infiltration, destroying the elasticity of the part and impeding the function of the cells invaded.

Both cellular and intercellullar elements may be infiltrated, though the first portion to be involved usually is the intercellular structure. Calcareous degeneration may be a final termination of fatty degeneration, and a very favorable one when vital organs, as the lungs, are involved, as, when this change has occurred, it becomes a permanent one, further degeneration and breaking down being arrested. In such cases the calcareous infiltration may be regarded as the lesser of two inevitable evils and welcome as a favorable termination of destructive action.

When the arteries become involved, much more serious consequences are liable to follow, as obstruction to these organs from inelasticity and narrowing of their lumen is certain to follow, with consequent destruction of parts supplied with blood through them. Atheromatous changes in the arteries are liable to be followed by this state, especially about the aorta and the arteries of the extremities, the middle coat, and finally the entire structure, becoming calcified. Senile gangrene is thus a common result of this condition.

Calcareous degeneration may occur under two different circumstances or influences. In the one there appears to be a perversion of the plastic forces, by which the normal calcareous elements of bone are deposited in an aberrant manner, or else there is an excess of calcareous material in the blood, many organs being simultaneously involved, such as the kidneys, lungs, stomach, intestines, dura mater, and liver. This may occur in osteomalacia, where there is an insufficient amount of lime in the bones to render them normally firm. In the other condition there has been previous disease of the parts infiltrated, the degeneration being a passive process due to the inactivity of the function-elements of affected parts. This form is that usually occurring in senile subjects.

The infiltration usually consists of carbonates and phosphates of calcium, with a small quantity of the magnesium salts. When treated with dilute mineral acids, there is bubbling of gases at first, and final dissolution of the calcareous material, the part regaining its elasticity.

Pigmentation.

Infiltration of the tissues with hæmoglobin causes more or less marked staining, which has been classed among the degenerations under the name of "pigmentary degeneration," when the hæmoglobin becomes converted into hæmatoidin, and is permanently fixed. Many cases of extravasation of blood are followed by a temporary staining of the tissues, but the coloring material is in a condition to be absorbed, and is soon taken up and carried away. But the hæmoglobin remains, in other cases, and becomes converted

into a granular or crystalline substance, hæmatoidin, which remains permanently fixed, staining the tissues.

Pigmentation, of itself, is not a serious condition; and when it affects the skin, it is a sign that some local or constitutional disturbance has been at work to bring about destruction of the red blood-corpuscles and liberation of their coloring material. Certain cachectic states are especially prone to be followed by permanent pigmentation of some of the tissues. Chronic malaria is one of these, and the fairest complexion is liable to be permanently browned in spots and sallowed, after a prolonged attack of this disease.

The tissues of the liver, kidneys, stomach, and other internal organs may be thus infiltrated after chronic atrophic local affections of these parts, the organs assuming a darkened color, and thus remaining during the remainder of life, though such diseases are usually fatal, not because the pigmentation is a serious matter, but because the causes which brought it about have also conspired to effect serious degenerative changes in vital organs.

The coloring material observed in lung-tissue is not usually of this character, as here minute particles of carbon are inhaled from the atmosphere and taken up by the cells of the mucous membrane lining the respiratory passages, and there permanently fixed. This has been termed a normal staining by some, and it would seem that this is proper, seeing that it is universally present in healthy lungs. However, without combustion of wood or coal these particles would not be present in the air, and the lungs would probably be free from the coloring matter. Sometimes this coloring is accompanied by true pigmentation, the result of inflammatory extravasation and other agencies, tending to fixation of hæmatoidin in the pulmonary tissues.

Staining of the tissues observed in subjects who have taken nitrate of silver for a long time is due to the deposit of particles of silver, and not to true pigmentation.

Necrosis.

Necrosis is the term applied to local death of animal tissues. This may occur from a variety of causes, and under a wide diversity of conditions. Whether it affects a single cell, group of cells, or an entire organ, complete suspension of nutrition and function follows, and the necrosed tissue begins a process of permanent dissolution and disorganization.

If the amount of necrosed tissue be small, and recuperative processes active, the disintegration may be molecular, the dead material being gradually absorbed and its place supplied by normal structure. When somewhat more extensive and the destruction more rapid, the breaking down may result in excavations, which are

afterward filled with fibrous or cicatricial tissue. When an extensive area of external soft tissue is involved, an obvious mass of darkened tissue—stained by infiltration with liberated hæmoglobin—appears, and is finally separated in a mass, the necrosed portion constituting a *sphacelus*, or *slough*, the condition being commonly known as 'mortification." If a mass of bone-tissue be involved instead, the separated portion is termed a *sequestrum*. Other masses seated deeply in certain tissues may be converted into cheesy-appearing bodies and become encapsulated, the condition being defined as *caseation;* or it may become infiltrated with calcareous material, *calcification*. When caseation or calcification occurs, the condition may be considered one of more or less pronounced permanency, though destructive changes may occur later.

Again, the dead tissue may be absorbed, leaving a cavity which becomes walled about with fibrous tissue, affording a space into which fluids infiltrate, the arrangement constituting a *cyst*. This is most liable to occur when areas of necrosis are located in the brain.

A peculiar form of necrosis is that which is termed *hyaline* or *coagulation* necrosis. This consists of the infiltration of necrosed masses with lymph, in which the third corpuscle has become liquefied, fibrinogen being developed, which ultimately welds the mass firmly together, a further change converting the necrosed area into hyaline material. This occurs in diphtheria, waxy degeneration of muscles, typhoid, typhus, relapsing fevers, tubercle, etc.

Colliquative necrosis is allied to the coagulative form, though it occurs in non-inflammatory conditions, in which the presence of scant amount of fibrinogen precludes coagulation, the affected part undergoing disintegration and liquefaction, instead of becoming coagulated. The brain contains little coagulable material, and colliquative necrosis is liable to occur here as a result of deficient supply of nutrition. Softening of the walls of the heart may also be due to colliquative necrosis when previous fatty degeneration has destroyed the muscular structure to great extent, necrosis finally being due to continued diminishing of the supply of blood resulting from increasing obstruction of the coronary arteries.

The *causes of necrosis* may be divided into two classes: (1) *Local injuries*, and (2) *arrest of nutrition.*

Local injuries may be due to mechanical or chemical causes; to inflammatory action; to long-continued febrile action inducing extreme cloudy degeneration in internal structures; to the poisonous influence of microbes, as in erysipelas, diphtheria, hospital gangrene, etc.

Arrest of nutrition is due to obstruction of the circulation from various causes, such as strangulation; gradual occlusion of arteries

from senile changes; pressure from new growths; embolism, and thrombosis.

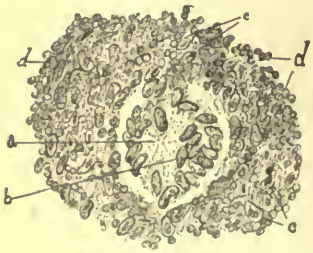
Dry gangrene is usually the result of necrosis in parts exposed to the air where the supplying arteries are occluded. The arterial obstruction may be the result of frost-bite, of a gradual filling of the lumen from senile changes in the arterial walls, such as calcification, atheroma, or of embolic infarction. The affected part becomes dark and leathery, then hard, black, and brittle. The anatomical elements, seen under the microscope, appear shrunken and withered.

Moist gangrène or sphacelus is mortification followed by putrefaction, and development of gases in the affected part. This change is due to the action of certain bacteria in the fluids of the gangrenous structure, which gain access either through the air or circulation. Increase of swelling, external blistering, offensive odors, oozing of putrefactive fluids, and disintegration of soft parts thus affected, follow at an early period. When bone becomes necrosed, the process of disintegration is more gradual.

Tuberculosis.

Tuberculosis is a condition characterized by the formation of nodules or tubercles within various tissues of the body, formed by an aggregation of cells, due to proliferation of surrounding tissue-elements, resulting from chronic inflammation caused by the presence of the tubercle bacilli. They are new growths, of low vitality.

Tubercles arise from small cells which originate from a mesoblastic membrane developed from connective tissue, or from the endothelium of blood-vessels or lymphatics. A focus of irritation is set up by the action of the bacilli upon the tissues, and proliferation and aggregation of these elements result. Leucocytes congregate about this focus, and granulation tissue is developed. Usually one or more epithelioid or lymphoid cells take on a hyper-plastic growth, and, fed by broken-up leucocytes, develop into giant cells, with homogeneous structure, numerous nuclei, and branching processes. About these are grouped epithelioid cells and leucocytes. Binding the tubercle in a mass is usually a fibrous reticulum, composed of the remains of normal connective tissue which has been distended and attenuated by the proliferated cells, and the branching processes of giant cells. In the meshes of this reticulum are the epithelioid and lymphoid cells, and leucocytes. An elementary tubercle is microscopic in size,



Miliary Tubercle.

a, Giant cell.
b, Nuclei of giant cell.
c, Epithelioid cells.
d, Lymphoid cells.

but numbers of these become aggregated into larger masses, visible to the naked eye. The tubercle formation is non-vascular, and when vessels are found in them they are the remains of preëxisting capillaries, around which the nodulated growth has developed. Bacilli are distributed throughout these growths, being most numerous about the central portion, where breaking down of structure begins. They are also found within the giant cells.

Tubercles are unstable structures, and they soon undergo fatty degeneration in the center from lack of nourishment, their non-vascular character precluding possibility of blood supply to interior parts. Consequently, the central part undergoes caseation, and, as softening proceeds, an abscess-cavity is formed in the center, and several miliary tubercles uniting, these cavities coalesce, forming considerable of an opening, the walls of which are studded with bacilli. Destruction of surrounding tissues results, and as this is progressive, destruction of the entire organ invaded follows, as well as of other parts to which the bacilli are distributed by the circulation. General destruction and dissolution of the entire organism must therefore finally result.

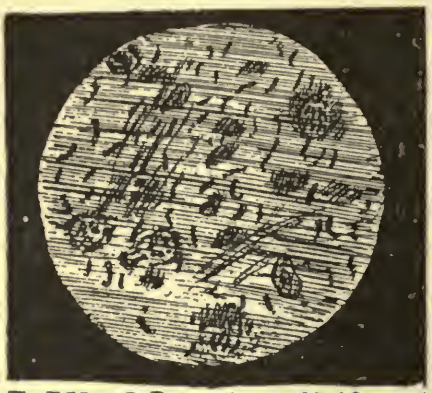
Another change which tubercles may undergo is that of fibrous degeneration, the common ending of proliferation of connective-tissue cells during chronic inflammation. Here the tissues which surround the tubercle take on fibrous inflammation, and the newly-developed fibrous tissue contracts and converts the growths into a fibrous nodule.

Tuberculosis is characterized by the subsequent invasion of various organs or the entire system, after the process has once begun. The bacilli enter the lymphatics, usually making halts here for a time, but they finally reach the blood through the thoracic duct, and are distributed throughout the entire system. In some cases they spread along membranes or through structures, invading new parts by continuity.

Tuberculization of tissue seldom occurs without complication. Many other morbid products are observable in tissues affected by tuberculosis, beside the typical tubercle structure. The general constitutional depravity results in feeble digestive power and nutrition, tending to atrophy of all the tissues, especially the muscles. The atrophy may end in degenerative change, and fatty metamorphosis and infiltration be one of the consequences. Inflammatory changes may develop pigmentary degeneration, hyaline necrosis, and cloudy swelling of epithelial elements. Tuberculous material may become fatty, calcareous, or fibrous. Hyperplasia of connective tissue may invade extensive areas.

Rapid loss of flesh and strength, involving all the functions, vegetative as well as volitional, acceleration of the pulse to a hundred beats per minute or near there, persistent elevation of the temperature of the body two or three degrees, hectic fever with colliquative sweats, and prominent evidence of local irritation corresponding to the part principally affected, are the leading clinical features of the condition.

The *tubercle bacilli* are vegetable microörganisms about one-third the diameter of a red blood-corpuscle in length, and about one-sixth as broad as long. They exist in both an active and passive condition, rapid multiplication characterizing their active state, while it is not unlikely that they may remain passive in the system for a long time, when conditions unfavorable to their increase are present, without producing any disturbance of health. Indeed, it seems as though it would be almost impossible for any one to escape contamination from them under such favoring circumstances as those which have formerly existed. When they are active, they present a beaded appearance, indicating the formation of spores, and rapid multiplication. They possess no power of spontaneous motion. They differ from most other bacteria in resisting the bleaching influence of strong mineral acids, after being colored.



TUBERCLE BACILLI - WITH SPORES IN SPUTUM. - SPECIMEN BY O. L. JONES, M.D.

Various *modes of infection* are known to exist. *Hereditary* or *congenital* tuberculosis occasionally occurs, though the disease is rarely thus transmitted. In such cases, the bacilli or their spores pass from the maternal circulation, or are transmitted by the male parent. It is probable that both avenues afford passage to the bacilli occasionally, as they are often found in the blood, and have been found in the testes.

Acquired tuberculosis may result from (1) *inhalation;* (2) *inoculation;* and (3) from *ingestion of tuberculous food.*

Inhalation of the bacilli is the usual mode of transmission, as the large proportion of pulmonary tuberculous disease attests. The facts that so many tuberculous subjects are able to be about until a short time before death, and that they have been encouraged to remain in the open air during pleasant weather, expectorating upon

the ground, where the sputum dries and becomes powdered dust, and the bacilli retaining their vitality for a long time, to be wafted here there in the atmosphere and inhaled by chance passers-by, account for the readiness with which this disease becomes spread about. Cloisters, prisons, and asylums are especially liable to become thus infected, dust deposited upon furniture, ceilings, and casements, being likely to become contaminated, long use of such quarters by a variety of persons being almost certain to result in the dissemination of tubercle bacilli by some one. Such places then become permanant hotbeds of infection, unless especial effort is made to thoroughly disinfect them, and afterwards confine tuberculous cases to special quarters. Private houses where the disease has prevailed are subject to the same danger.

It is patent that careful and rigorous measures to collect and thoroughly destroy the sputum of tuberculous patients will be the most successful method of arresting the ravages of the disease.

Inoculation is another common method of transmission. This may be developed through the act of kissing, from the use of contaminated surgical instruments, the hypodermic syringe, dental forceps, etc. Butchers may be inoculated from the flesh or skin of bovines, for the disease is common among horned domestic cattle. Handling contaminated meat is also liable to result in inoculation before cooking, though long-continued heat destroys the bacilli. Medical students and practitioners are liable to inoculation during dissection and autopsies of tuberculous persons, as well as from operations upon such subjects. It is asserted that the disease has been disseminated among Jewish children from the practice of sucking the fresh wound of the prepuce by tuberculous operators.

Infection from food often occurs. One of the most widespread causes of infection is the use of cows' milk in feeding infants. It is conceded that a large percentage of cattle are tuberculous. Much of the condensed milk, as well as that furnished by dairymen, is therefore subject to suspicion of tuberculous infection. Acute miliary tuberculosis, a disease very common among children, is in all probability the result of such a diet, the alimentary canal offering a ready place for the introduction and dissemination of the bacilli. A diet of raw meat, such as German sausage and raw beef, is also inimical to safety on this score. Danger lurks in every piece of raw beef as an article of food, though a person may make occasional use of it for a lifetime and escape infection.

The treatment of tuberculosis has proven almost a complete failure from time immemorial. And recent times, despite the remarkable advances which have been made in the knowledge of its etiology,

have afforded us little improvement upon treatment. In spite of the splendid achievements of Koch, in the discovery of the tubercle bacilli, his tuberculin inoculations have signally failed to offer any hope of beneficial results. And it is probable that other researches in this direction will prove as fruitless.

The majority of successful innovations in medicine have sprung from humble sources. Savants have proven failures, when the successful treatment of stubborn diseases has occupied their attention. Usually their reasoning has been too crude for the intricate processes of life and correspondence with physiological principles. Attempt to cure tuberculosis by directing the means toward the extermination of the bacilli in the body, will probably always prove a failure. Changes of climate, whereby the normal forces are encouraged, and affected organs placed under more favorable circumstances, are probably the best measures adopted by the profession at the present time, though I believe there exist more positive measures, which will cure a large majority of cases of pulmonary consumption, after such an advance has been made that numerous pulmonary hemorrhages have occurred, if faithfully employed. In order to prepare the reader for a proper conception of these means, I will transcribe the account of

THE DUKE OF WURTEMBERG'S REMARKABLE CURE BY JOHANNES SCHROTH,

as translated from the German by Dr. William Weber, an alumnus of the California Medical College, and former patient and pupil of Schroth. Though not literal in every particular, the following contains the gist of this translation:

"It is incredible how few people there are who think for themselves, and how often the seemingly most original and independent persons are found, upon close investigation, to be only the slaves of the thoughts and opinions of others. Enough . . . the sluices of prevalent opinion were opened against the method of Schroth, and only the great importance of its new and unheard-of ideas could prevent the same from being consigned to oblivion. At that time cases were reported, such, for instance, as that of the Duke of Wurtemberg, which, from the importance of the person involved, commanded attention. I shall here give some details of that cure, which will furnish some of the characteristics of Schroth, as well as a better understanding of his mode of treatment.

"Duke William of Wurtemberg, Captain of the Royal Imperial Austrian Regiment of the Line, No. 45, in storming a redoubt of the enemy at the battle near Novara (Italy), on March 12th, 1849, at about noon-time, was wounded one inch below the patella, by a shot fired at close range—a pointed bullet. This injured the tendons

and ligaments of the knee-joint, perforated and splintered the tibia, separated the muscles of the calf, and severed several arteries, coming out on the posterior surface of the leg.

"The Duke, wounded in this manner, lay on the battle field for some time, and later was removed to a field-hospital, and the following day, to Mortara, near the conquered redoubt. The loss of blood was considerable, but the weakness caused thereby rendered him less sensitive to pain. Nevertheless, at the hospital, the Duke was bled twice.

"On March 28th, he was transported to Pavia, and suffered great pain during the trip. Suppuration had set in, which spread over the whole calf. Compressing bandages sometimes increased the pain to the point of syncope. Pyrexia set in, and, in the opinion of his attending physician, his life was in imminent jeopardy.

"On account of the malarious climate, the Duke was removed to Mailand, and was here taken charge of by the Surgeon in Chief of the Royal Imperial Lombard Gendarmerie Regiment, the latter part of April. On May 7th, a long incision was made in the calf to arrest the progress of fistulæ in that region, and a progressive improvement seemed to follow, for a short time. About the middle of June the patient left his bed for the first time, but could make no use of his leg. Warm baths were administered to him every other day, and he attempted to walk on crutches, and occasionally rode about in a carriage, toward evening.

"Evidently, however, there was little improvement for the following six weeks, for the pain increased, and the Duke left Mailand the first of August en route for Baden Springs, near Vienna, for the purpose of trying the effect of these waters for relief. His condition was now so extreme that convulsions occasionally occurred. Though this trip was signalized by excruciating suffering, he improved somewhat at Baden for the first fortnight, but after the effects of the change passed away, his strength again began to fail, and the inflammation and pain in the diseased leg continued to increase with added suffering. Up to this time the patient had never been able to stand on his diseased leg. As in Italy, prominent surgeons at Vienna advised him to submit to amputation, but this was obstinately opposed by the Duke.

"His condition becoming continually worse, he went to Karlsruhe, in Prussian Upper Silesia (the place of his birth), arriving there September 22d, very much exhausted. An eminent surgeon was now summoned from Berlin, who proposed to remove the head of the tibia; but as the Duke was very low, and as the surgeon could not positively promise a satisfactory result, the proposal was rejected.

"At this desperate stage of affairs, at the advice of his former teacher, Dr. Merten, and against the wishes of his family, the Prince decided to go and consult the naturalist physician, Johannes Schroth, at Lindewiese, near Gräfenberg, whose reputation for wonderful success in the treatment of similar troubles had long been noised abroad. On November 12th, he therefore left for Lindeweise, arriving in an exhausted condition on the following 14th.

"When Schroth investigated the case, he was appalled by the appearance of the diseased leg. The knee was swollen to half more than its normal size, and the swelling was hardened by hyperplastic deposits in the inflamed structures and about the fistulous openings. The part was extremely sensitive and painful, and the least motion of the joint was impossible. The bone was enlarged, and almost the entire tibia was found honeycombed. The probe would break through the decomposed and softened bone at almost every point along its shaft and about the tuberosities, with the most gentle pressure. At a depth of about three inches from the surface, in the neighborhood of the old wound, were splinters of bone, and from here fetid ichor was constantly discharged. From above the knee down into the calf, enlarged and painful lymphatic glands (the size of a pigeon's egg) were found. Continuing with the examination Schroth found the liver and spleen enlarged: a condition he ascribed to laguna fever, from which the patient had suffered while in Venice.

"The difficulty was, not only to cure the local affection, but to invigorate, if possible, the broken-down constitution. To do this Schroth thought it necessary that an excretion of morbid matter from the system should take place, and formation of new and healthy blood be induced. It was his idea that only under such circumstances could the diseased organs be regenerated to normal conditions. The physician present, educated in the principles of the scientific schools, could not believe in a regular treatment without remedies, and remonstrated with Schroth, advising him not to interfere with such a desperate case, for fear that the apparently unhappy final result would militate against him. But Schroth asked of the Duke three days for consideration, and then said, with full conviction, to the well-meaning physician: 'The Duke will be cured. I am sure of success.'

"The discoverer of this mode of treatment, to make such a cure possible, first found it necessary to build up the entire constitution. Otherwise a continuous curative reaction would have been impossible. About the middle of November, Schroth commenced the treatment, and from this time until December 30th, the Duke ate and drank every day. Nights, he was wrapped in a peculiar abdominal

pack, invented by Schroth. The diseased leg was gently rubbed mornings and evenings, with the moistened hand, as long as possible on account of pain excited, then soft linen, in the form of straps, was wetted in cold water and wrapped about it fourteen or sixteen thicknesses, and allowed to remain twelve hours, by which time the dressing had become dry. The Prince himself thus reports the history of the treatment:

"'My diet was extremely simple. In the forenoon I ate nothing, as I did not like the stale bread. At noon I got a piece of dry boiled beef, and occasionally, some dry rice or potatoes with the same in the afternoon, at about four o'clock, and was permitted to drink some wine, to which, however, I could not accustom myself, in spite of being very thirsty. Not until after a fortnight I began to get accustomed to get along without water. The success of this treatment, which was just as simple as ingenious, was surprising in the highest degree. On the second day the already cicatricial portions of the wound opened again, and a great deal of pus was discharged; the existing severe pain diminished, I got easy, and the fever disappeared. At the same time my appetite improved, and at the fair at Lindeweise, which was just then celebrated, I ate as much as anyone. On this occasion I tested Old Schroth's ingenuity. To my great satisfaction he allowed me to eat some beef soup, which he had strictly forbidden in case of wounds. On the very same evening, when the bandage was removed, we noticed a very bad swelling about the knee, just as he had predicted, and Schroth explained to me the effect of beef soup on wounds, as long as the stomach cannot digest normally. In order to have the case tested further, he encouraged me to drink some cold beer, on the next afternoon. I drank about two glasses, and when he bandaged the wound about three hours afterwards, it showed a gray-colored, morbid pus, on the lower part of the wound, and on the upper, watery matter was discharged. The borders of the wound were also very red and painful. This was a clear proof that my digestion was very poor, and that all fluids went directly to the wound. A general consumption would have been the inevitable consequence of persisting in such a course.'

"On December 2d, the patient could step on the wounded leg for the first time since Novara, and walk several times up and down his room. The limb showed more strength, but there was yet pain in the knee and articulations of the foot. At last, the fever left entirely.

"When the healing process commenced, the regular treatment was begun. He was now wrapped in large packs, but his system was so much exhausted that Schroth allowed him to continue his previous

diet. Not until the middle of January was his strength built up sufficiently to enable him to undergo the main or regular treatment, and the patient himself says the following about it:

"'The more thirsty I was, the more pus was discharged from the wound. This was a dark, tenacious liquid, mixed with blood, and of a very bad odor. The more pus discharged, the stronger and more movable became the leg. When, after about three weeks, my tongue got clean, and the discharged liquid became lighter in color, Schroth allowed me to enter the so-called after-cure. In a few days the suppuration ceased, and in a few days more the wounds closed up entirely. During the main cure I had lost a great deal of flesh, but now, when I was filling up again, my leg got very strong, and it took only a short time to overcome the limping and be again in full strength and health. At the end of January I made my appearance at Gräfenberg, to show the followers of Priesnitz' hydropathic cure the great and wonderful results of Schroth's mode of treatment. On March 1st I considered myself as being in normal health, and am under obligation to Father Schroth for this extraordinary cure in the short space of sixteen weeks.'

"The Duke, in grateful acknowledgment, published the following article, in No. 43 of the journal *Oesterreichischer Soldatenfreund*, Vienna, April 23d, 1850:

"'To my Comrades in the Army: The undersigned considers it his duty to direct the attention of his wounded comrades to a new mode of treatment, which effects a surer cure than all modes of treatment practiced by physicians. The farmer, Johannes Schroth, at Neiderlindewiese, near Freiwaldau, in Austrian Silesia, for many years has treated fresh and old wounds by a new and extraordinary method, and at all times attains a most successful result, but the same is very little known.

"'A great many call his cure "the stale bread cure," and ridicule the same, because they think he cures wounds with stale bread; but this is not the case. The principle of Schroth's cure is not to allow much fluid to go to the wounds, and he effects it by a strict, dry diet. He forbids his patients the drinking of water entirely, and orders mainly stale bread and wine for nourishment. At the same time he applies local packs, which may remain a longer or shorter time, depending upon circumstances. It cannot be said that the cure is easy, but it is neither very hard, and not a great sacrifice for a sure recovery of health.

"'I will cite some cases, beginning with my own. A pointed ball had pierced my shin directly below the knee. I had been in bed for

nine months, and there was no hope of a recovery; but Schroth cured me in four months. The pensioned Colonel of Tschebury had been suffering since 1809, from the consequences of several wounds. During 1849 he used the cure and got cured from all his old troubles. An old wound of course needs a longer time to get well than a new one, but in the latter case the success was extraordinarily rapid and brilliant.

"'Two cases more which occurred under my own observation will finish my account of cures. A farmer's girl had her arm fractured and the joint splintered. By the use of packs and a peculiar diet, Schroth cured this case in such a manner that she could afterward use her arm as well as ever. An old, strong farmer cut his shin-bone with an ax, almost through, and several tendons were severed. After three weeks' treatment he was able to use his leg again.

"'I shall be very glad at any time to give my comrades of the army details of this treatment, which saved me, and, I hope, will do a great deal more for others.

WILLIAM, Duke of Wurtemberg.

"'Vienna, August. 1850.'"

It is upon such principles as these that Dr. Weber depends for the cure of pulmonary and other forms of tuberculosis; and that he cures, I have reason to know. Withdrawal of water from the system increases the proportion of red corpuscles and other solid constituents and improves the reparative power of the circulating fluids. Withdrawal of fluids also seems to cause rapid destruction of abnormal tissues of feeble vitality, and they melt away and are discharged. Tubercles break down and are cast off under the new conditions, while the formation of granulation-tissue and other steps of tubercle growth are arrested, and the evacuated cavities are cicatrized.

There are many unpleasant features connected with the management of this mode of treatment of such cases, the principal causes of contention being the prejudices of both popular and professional sentiment and education. The idea of limiting the diet of a consumptive patient, in the light of our present education, seems atrocious; yet we might recollect that those who are most freely fed may die sooner than those who are not so well favored (?). Plenty of ordinary food and drink seem to furnish the very pabulum required for the rapid development of the bacilli and production of tubercle deposit.

Wine alleviates the thirst somewhat, but not altogether; but here it is a medicine. It hardens the tissues, and fortifies them against the inroads of the bacilli. Possibly these are starved out for lack of water, as vegetable organisms require this for their proper growth, and animal tissues possessing the best absorbing power, probably

and animal tissues possessing the best absorbing power, probably rob them of the limited supply furnished by a dry diet. But these are theories, though the facts remain.

The patient becomes fearfully emaciated at first, and had better not be seen by his friends, as they will not now be likely to add encouragement to his resolution to persevere. As 75 per cent of the body is water, withdrawal of a large proportion of the amount ordinarily consumed must bring about a remarkable shrinkage in bulk. The patient soon becomes fearfully weak and emaciated, the expectoration increases in amount, and symptoms are at first most threatening. But after a few weeks the amount of material expectorated begins to grow markedly less in amount, and finally ceases altogether, though treatment must be faithfully continued until the cough and expectoration have ceased entirely. Then, even upon the limited diet, the patient gradually regains digestive power and strength.

IV. BACTERIOLOGY.

Bacteriology is the science of bacteria. Bacteria, microörganisms, or microbes, are minute vegetable organisms representing the lower forms of vegetable life and related to the algæ botanically, which naturally maintain a parasitic existence in human and animal fluids and tissues, but many of which may be cultivated artificially outside these situations. As vegetable organisms, they are peculiar on account of the absence of chlorophyl in their composition, and in their mode of reproduction by fission, and the formation of spores. They normally exist in communities of many different kinds, and possess the power of rapid multiplication, under proper circumstances. This fact confused positive knowledge of their relation to disease until bacteriological research separated them by cultivation into pure cultures, enabling inocultion tests as to the specific character of many distinct forms, to be made upon animals.

When developed, a bacterium represents a cell, consisting of an enveloping membrane containing protoplasm. The membrane is usually very firm and adherent, is separated from the protoplasm with difficulty, and is also resistant to external influences.

TECHNOLOGY.

A knowledge of the peculiar characteristics of individual bacteria comes from ability to isolate them by cultivation. A portion of diseased structure may contain numerous colonies of bacteria in confused admixture, these differing materially in their properties and significance. One variety may be innocuous, another may exist there accidentally, while still others may sustain a direct causal relation to the pathological condition.

Nutrient media are necessary for the cultivation of bacteria outside of animal tissues; and of these two classes are employed, viz., *natural* and *artificial.*

Natural media are those which are employed in their natural state, such as pleuritic fluid, the fluid of hydrocele, blood-serum, potato and other vegetables, eggs, etc.

Artificial media are prepared substances, such as bouillon, gelatine, agar, and certain saline mixtures, such as Pasteur's, Cohn's, and other preparations classed as *mineral media.*

Some bacteria thrive best on one material, and others on another.

A variety of media are therefore necessary, in order to meet the demands of different occasions.

Sterilization of everything connected with the manipulation of bacteria is necessary in the successful propagation of pure cultures. Great dexterity is also requisite upon the part of the manipulator, in order that successful results may be arrived at. A sterilized chamber for the prosecution of bacteriological experiments is essential, and ovens for the sterilization of everything connected with the work must be at hand, as a few seconds' exposure to the air may contaminate a pure culture, and thus destroy accuracy. On this account the most expert operator may fail to propagate successfully.

A knowledge of *microscopy* is also essential, and the operator must understand mounting specimens and adjusting them upon the stage, so as to bring them into proper light and focus.

Staining of specimens is also an important matter, and this comprehends considerable skill and experience.

The *technique* of obtaining a *pure culture* from a mixture of a variety of germs may be briefly summarized as follows:

Sterilized media are inoculated with a sterilized platinum needle from the pathological specimen to be investigated, in successive series, upon the principle of dilution observed in preparing drug-attenuations. For example, after one inoculation has been made the second is carried on from this, the third from the second, and so on. Usually the third inoculation so reduces the number of bacteria that they may be singled out by spreading the medium in a thin layer upon a sterilized plate, each one then breeding a separate colony. Each of these may now be transferred to a separate culture-medium, and if it contain only the one kind of microbes, upon growing, it constitutes a *pure culture.*

Guinea-pigs, mice, and other lower animals may be inoculated from these cultures, and the effects observed. Some may prove wholly inocuous, others producing variable symptoms. When one of the cultures constantly proves toxic, its effects are more carefully

observed, and in this manner the specific causes of various infectious diseases are determined. However, there is not always a certainty as to the specific microbe of a disease, as bacteriologists are frequently compelled to abandon positions which have been taken with much positiveness. The toxines generated by bacteria seem to vary in their poisonous influence in different individuals, depending perhaps upon constitutional susceptibility or temporary predisposition, owing to constitutional depravity, and confusion arises, where it might seem that like causes ought always to be followed by like results. To illustrate the meaning here it may be remarked that the Klebs-Löffler bacillus, which was but recently regarded as the specific microörganism of diphtheria—declared so by both investigators after careful and painstaking investigation—seems innocuous in some instances, and has been found in the buccal and nasal cavities of perfectly healthy persons. With all the positiveness of bacterial research, then, there seems to be much uncertainty, after all.

A large majority of bacterial forms grow freely in gelatine and agar, though some, such as the tubercle bacillus, gonococcus, and others, require special media. Others, such, for example, as the spirochæte of Obermaier, cannot be cultivated in any known medium.

STAINING AND PREPARING.

THE peculiar resistant qualities of bacteria enable them to retain coloring material when surrounding tissues or media yield to bleaching influences. Aniline colors are chiefly employed for staining, these being retained by the microbes, while they are removed from surrounding parts by bleaching, with different processes. Thus the bacteria are left highly colored, while the field in which they lie is approximately achromatic, rendering them prominent to vision under proper magnifying power. Basic dyes possess special value for penetrating the nuclei of cells and bacteria, and when such colored specimens are heated in acetic acid the nuclei are dissolved, diffusing the coloring material through the protoplasm generally, rendering the complete organism distinct. The basic dyes most in use are methyl blue, methyl violet, gentian violet, dahlia, basic fuchsine, Bismarck brown, etc. Some bacteria cannot be penetrated with simple solutions of basic dyes, and more complex preparations are required.

Löffler's solution may be resorted to here. A compound of 30 parts of a concentrated alcoholic solution of methyl blue, and 100 parts of a 1-1000 solution of caustic potash in water is first used. After the preparations remain in this for a few minutes, they should be treated with a 1% solution of acetic acid, then washed with absolute alcohol, and cleared with cedar oil.

Gram's method is a favorite one with bacteriologists. In this, the specimens must be put to macerate in absolute alcohol, before the staining process. After removal from this, the prepared cover-glasses are allowed to float, prepared side down, upon a mixture composed of water solution of aniline oil 100 parts, to saturated solution of gentian violet 5 parts, for a few minutes. From here they are transferred to an iodo-iodide of potash solution, and allowed to remain for one minute, when they are washed with absolute alcohol from one to three minutes, until they become free from color to the naked eye. They are then cleaned up in oil of cloves, and mounted in Canada balsam or glycerine-jelly.

This method stains the bacillus tuberculosis; bacillus anthracis; bacillus lepræ; diplococcus pneumoniæ; pneumococcus Friedlander; streptococcus erysipelatis; actinomyces; and all pyogenic bacteria.

It does not stain the bacillus of glanders (bacillus mallei); bacillus typhosus (bacillus of Eberth); diplococcus intercellularis meningitis; gonococcus; spirillum choleræ asiaticæ (comma bacillus); or spirochæte Obermaieri (relapsing fever).

Of these, Löffler's solution stains gonococcus, diplococcus intercellularis, and bacillus typhosus. Water solutions of dyes stain bacillus mallei, spirillum choleræ, and spirochæte Obermaieri, these being afterward brought out with 1% solution of acetic acid.

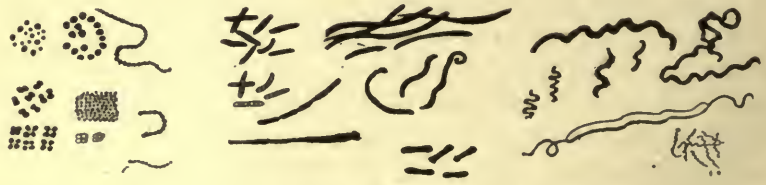
Preparations may be double-stained, so that the field and bacteria present contrast colors, the bacteria presenting a deep blue tint, while the ground or field is carmine, a strong and attractive contrast thus being made, and the bacteria being brought out more clearly.

The tubercle bacillus is so important that special attention to its staining will be proper. In common with the bacillus of leprosy it differs from other bacteria in resisting the bleaching power of strong mineral acids. Cover-glass preparations of sputum, blood, tubercle, pus, etc., containing this microbe should be first treated with a solution composed of 100 parts of aniline water, 11 parts of an alcoholic solution of fuchsine, methylene blue, or methyl violet, and 10 parts of absolute alcohol (Erlich's solution). At ordinary temperatures the specimens should be allowed to remain in this for twenty-four hours. If the solution containing the specimens is heated, a few minutes will suffice, this being termed the "rapid process." The cover-glass preparations should now be removed and passed over a gas-jet three or four times, until steam begins to rise. After this they should be washed in a solution consisting of nitric acid one part, to water two or three parts. After nearly decolorized, wash in plenty of water or alcohol. Sections should be treated with alcohol, cleared with oil of cloves, and preserved in balsam.

But this process will also develop the bacillus of leprosy, and the following method will be useful to distinguish between these two forms of bacteria. Fill a watch-glass with water, and add from four to six drops of a saturated alcoholic solution of fuchsine. In this place the cover-glass preparation for six or eight minutes, and afterward treat with 1-10% nitric acid for twenty seconds, afterward washing in distilled water and treating with aqueous solution of methylene blue. After this clear with oil of cloves. This removes coloring from the tubercle bacilli, leaving only the bacilli of leprosy. If nothing can now be seen, the microbes before observed were evidently those of tuberculosis. If sections are to be treated, allow them to remain in Erlich's solution for two or three minutes, after which pass through the nitric acid solution for half a minute, then stain with water solution of methyl blue. Only the bacilli will be stained by this process.

BIOLOGY.

EARLY investigators in the domain of bacteria inclined toward the belief that they all sprung from a common source, and that the different forms represented various stages of development, or the influences of varying environment. But, while one species may be represented by different forms during development, the mature form of an individual species does not vary, and its individual characteristics and properties are always the same, under the same circumstances.



FORMS OF BACTERIA.

Experiments in propagation have demonstrated that more than thirty generations of pus-microbes and other kinds may be cultivated without change of form or property. They are as distinctly separate species at the end of this time as in the beginning.

Three distinct forms of pathogenic bacteria have been discovered and described, namely, (1) the ball or berry (*coccus*), (2) the rod (*bacillus*), and (3) the corkscrew (*spirillum* or *spirochæte*).

In some instances there may be a near approach in the resemblance between two individuals belonging to different classes. For

instance, an oblong coccus may resemble a short bacillus, a double coccus a short bacillus with club-shaped ends, etc.

When cocci are arranged in pairs, they are called diplococci. When in fours, and in the form of a square, micrococcus tetragones. When in the form of a cube, sarcina. When arranged in the shape of a bunch of grapes, staphylococci. When they are arranged in chains, streptococci. An irregular bunch, held together in a mass by a viscid fluid, is termed a zoöglœa.

When circumstances are favorable to their development, the *reproduction* of bacteria occurs with great rapidity. In some forms multiplication is brought about by fission or splitting of the bacterium into halves. In others, the multiplication results from the development of spores, similar to the seed formation of flowering plants. The cocci multiply by fission. A cell elongates and becomes constricted in the middle. As the constricting process continues, the bacterium separates into two equal parts, each of which soon attains the size of the parent cell. If the cells remain in contact and multiplication continues longitudinally, a chain or streptococcus is formed. Rod bacteria which multiply by fission separate in the middle and each half grows to the size of the parent, when separation occurs in each half, as in the parent cell. Bacilli usually multiply from spores. When these develop in a bacillus, darkened spots first appear at equidistant points, which soon (within twenty hours) develop into pearly, opaque segments. These then part, and each develops into a separate bacterium. Spores are usually more resisting to destructive influence than bacteria, though a heat of 212° F. destroys them.

The *nutrition* of bacteria demands oxygen, nitrogen, carbon, water, and a limited amount of mineral salts. Water is indispensable to their growth, and prolonged desiccation is fatal to many, though others may exist for a long time in a dormant state when desiccated, to renew activity when moisture is supplied. Oxygen is indispensable, though some grow in open air, while others cannot be cultivated except in media in which they can grow beneath the surface. Bacteria which grow only in the open air are termed aërobii; those which only grow away from the open air, anaërobii; others, which can grow in either situation, are termed anaërobii by election. Bacteria obtain their carbon and nitrogen principally from the media in which they grow. Their growth brings about certain changes in the media which determine their character, in certain directions. For example, certain kinds decompose their media and produce color (chromogenic bacteria). Others give rise to ferments (zymogenic bacteria). Still others, and the important class to physicians, origi-

nate various toxic conditions (pathogenic or disease-producing bacteria).

Many bacteria possess the power of *spontaneous motion*, from conditions of their nutrition. The motion may be directly due to cilia, with which some forms are supplied, or, in other cases, to contraction of the protoplasm. In other instances both these agencies may be concerned.

Certain conditions or provisions are essential in determining the identity of *pathogenic bacteria*. They must be found in the bodies of animals or human subjects suffering with or dead from disease. They should be cultivated from such sources, when possible, and a pure culture of them should produce the same disease when inoculated into animals. Such animals, when diseased, should contain in their blood and tissues the identical bacteria found in the tissues or fluids of the affected individual in the beginning.

Bacteria *cause disease* in different ways. Some kinds (pyogenic bacteria) attack the leucocytes and embryonal cells and convert them into pus-corpuscles. Others produce hyperplasia and the development of new growth, as in the generation of tubercle. Still others generate toxines, which produce constitutional diseases, such as the infectious fevers.

Some infectious diseases have not yet been demonstrated to be the results of specific germs, these probably existing, but having thus far escaped the scrutiny of bacteriologists. Germs have been discovered and declared the causes of disease, and afterward demonstrated to be harmless saprophytes. The field of bacteriology is yet full of speculation and uncertainties. Much remains to be cleared up, and doubtless much has been accepted as true which later research will determine as unfounded.

A few bacteria have been pretty well located and described. Others are yet subjects of speculation, investigation, and debate. Those which are generally accepted as specific causes of disease will be briefly described in the following order:

PATHOGENIC COCCI.

Diplococcus Intercellularis Meningitis.—This is found in the exudation of cerebro-spinal meningitis. The cocci occur in pairs, united.

Diplococcus Pneumoniæ Lanceolatus.—Occurs in the exudation of croupous pneumonia. Cocci united in twos. Under cultivation, loses its capsule.

Gonococcus.—Occurs in gonorrhœal pus. The cocci occur in twos, similar to the arrangement of coffee-grains.

Staphylococcus Pyogenes Aureus.—Found in yellow pus. The cells are grouped in bunches, and are of a yellow gold color.

Staphylococcus Pyogenes Albus.—Found in pus, similar to preceding, except that the color of the cultures is white instead of yellow.

Staphylococcus Pyogenes Citrus.—Also found in pus. Resembles other pus-microbes, except that the cultures are lemon-colored.

Streptococcus Pyogenes.—Occurs in the pus of phlegmons, in the shape of chains of cocci.

Streptococcus Erysipelatis.—Found in the lymph-spaces of erysipelatous parts. It is probably identical with streptococcus pyogenes, though found under different circumstances.

PATHOGENIC BACILLI.

Bacillus Anthracis.—The bacillus of anthrax, found in the lymph and blood of animals suffering from splenic fever. It occurs in single rods or long chains, when cultivated.

Bacillus Œdematis Malignæ.—The bacillus of malignant œdema. Found in human subjects and animals suffering with this disease. Grows under gelatine (anærobic), giving rise to gas bubbles.

Bacillus Leprœ.—The microbe of leprosy, found in leprous tubercle. It resembles the bacillus of tuberculosis. Grows in blood-serum.

Bacillus Mallei.—A short, slender rod, resembling the bacillus of tubercle, found in the secretions and tissues of subjects suffering with glanders. Grows on blood-serum and potato.

Bacillus Pneumoniæ.—Short rod, single or in chains, found in the exudation of croupous pneumonia. It is covered with a capsule in pneumonia, but the capsule is absent in cultures. It grows on gelatine.

Bacillus Rhinosclerma.—Found in the tubercles of rhinosclerma. Resembles the diplococcus pneumoniæ in form and cultures.

Bacillus of Syphilis.—A short rod found in the lesions of syphilis. It has not been cultivated.

Bacillus Typhosus.—The bacillus of typhoid fever. A short rod with rounded ends, found in the evacuations, and also in the mesenteric glands and spleen of subjects affected with the disease.

PATHOGENIC SPIRILLI.

Spirillum Cholerœ Asiaticæ.—Curved rods, resembling a comma in shape, sometimes curved in the shape of a letter S, and sometims of corkscrew form. Found in the evacuations of cholera. Grows in gelatine.

Spirochæte Obermaieri.—The spirillum of relapsing fever. It has never been cultivated, but has been inoculated, and has reproduced the disease in healthy animals and men.

PATHOGENIC FUNGI.

Actinomyces.—The fungus found in the tumors of actinomycosis. A club-shaped fungus, which grows in the form of radii. Has been cultivated on agar, and grows in small grayish dots.

Achorion Schönleinii.—The fungus found in patches of favus. When cultivated upon agar, it grows in patches presenting the characteristic color of this affection.

But the science of bacteriology does not include all the micro-örganisms of disease. Bacteria are vegetable organisms, while there are numerous instances in which disease is caused by the presence of animalcules in the body. The plasmodium of malaria is an example of this kind, and the filaria sanguinis hominis another, these creatures existing in the blood, while the trichina spiralis, echinococcus, germ of the tapeworm, etc., are embedded in the solid tissues. Due notice will be given these parasites under the diseases in which they occur.

SECTION II.

SPECIFIC INFECTIOUS DISEASES.

I. TYPHOID FEVER.

Synonyms.—Enteric Fever; Typhus Abdominalis.

Definition.—An acute infectious disease excited by a specific bacillus, and marked by inflammation of Peyer's glands; clinically characterized by fever of gradual development, headache, delirium, stupor, abdominal distention, diarrhœa, splenic engorgement, and an abdominal rash.

Historical Note.—Prior to 1813, typhoid fever was not distinguished from other forms of protracted pyrexial disease. In 1813, Paul Bretonneau, of Tours, described "dothiénentérite" as a distinct disease, and contemporary writers described "entero-mesenteric fever." The views of Bretonneau were disseminated by his pupils, especially by Trousseau and Velpeau, until the profession of Paris accepted them. In 1829, Louis' work was published, containing a description of typhoid fever for the first time under that name in a recognized text-book. The students of Louis included members of various foreign nations, among whom were Americans, Gerhard, of Philadelphia, soon publishing, in the *American Journal of Medical Sciences*, the first full and accurate account of the disease ever written in any language. James Jackson, Jr., of Boston, another pupil of Louis, returned from Paris in 1833, and soon proved, in his father's hospital wards, the identity of the then so-called typhus fever of this country with typhoid. His death occurred soon afterwards, but in 1838 and 1839, James Jackson, Sr., and Enoch Hale prepared memoirs which were published by the Massachusetts General Hospital, fully describing the difference between typhus and typhoid fever, their views being generally accepted by American physicians, though it was several years before the mass of the profession in Europe admitted the distinction between these two forms of febrile disease. It was not until 1850 that all points of dispute were finally settled.

Etiology.—It is now generally conceded that typhoid fever is the result of an infection of the system of the affected individual with a specific germ (the bacillus of Eberth). This is found most constantly in the intestinal discharges of the sufferer, and, as it retains its vitality for a long time, it may finally drift into wells, reservoirs, or springs, thus contaminating drinking water, the infection entering by way of the alimentary canal. Even without previous

history of typhoid infection, the use of potable water from wells or springs located in the neighborhood of privies, sewers, or barn-yards is hazardous, as fecal material seems to possess the property of preserving the typhoid bacillus for years, if it be not a medium for the germ from some unknown source outside the human body. After the historical flood which occurred in Western Pennsylvania, in 1889, a widespread epidemic of typhoid fever occurred in the rural districts, where it had formerly been unknown for a long time, and the disease was traced to infected wells in most instances, where it seemed probable, from their location, that they had received washings from neighboring privies. But the recent fecal material of typhoid-fever patients is less virulent, though doubtless it is frequently a source of contamination among nurses and those who wash the clothing. In large towns and cities, where milk is distributed from common supplies and transported from rural districts where contaminated water has been used to rinse the cans, without scalding, and to increase the bulk of the article, the cause of epidemics of this disease is readily accounted for, provided the water supply of the hydrants has not been defiled. Doubtless the bacilli may sometimes enter the circulation through the lungs, in the condition of a dry powder, floating in the atmosphere.

Certain *predisposing* causes are believed to operate in encouraging the spread of epidemics, such, for example, as the autumn season, early life, etc. The greater prevalence of typhoid fever in the autumn is probably due to the fact that greater liability to contamination from drinking water and floating germs in the atmosphere then prevails. After a protracted drought, the ground water is low, and springs and other water sources drain contaminated foci closely, thus being more likely to be charged with the specific poison. The same atmospheric condition may result in the presence of floating germs in the air, which may fall into drinking water, or enter the system through the organs of respiration. *Youth* and early adult life is the period of greatest susceptibility, the majority of cases occurring between the ages of fifteen and twenty-five. It is progressively infrequent after thirty-five, though it may occur at any period, the fœtus becoming infected through the maternal circulation during late months of pregnancy. Not all who are exposed to the contagion suffer from it, as all are not susceptible, and some who are affected do not suffer severely, perambulating cases being frequently observed. It occurs both epidemically and endemically, being endemic in most large cities, in which case there is great difficulty in tracing the infection to its source.

One attack usually confers immunity against subsequent exposure.

The *bacillus of typhoid* fever is a short, thick, motile bacterium with rounded ends, in one and sometimes both of which an opaque glistening spot is observed, supposed by some to represent a spore, it being noticed most frequently in cultures. These bacilli may be preserved for an indefinite time in water, and here they probably slowly multiply. A heat of 140° F. destroys, though extreme cold does not injure them, congealment in ice producing no apparent injurious effect. They multiply rapidly in milk, and cultures grow in various other culture-media, the growth being invisible on potato. Repeated trials of inoculation upon animals have failed to produce the disease, though this has been explained by the hypothesis that animals are not susceptible.

Pathology.—It is highly probable that the constitutional disturbances arise from toxines generated by the bacilli. Brieger has described a typhotoxine, and Fränkel a toxalbumin, though knowledge of these poisons is not yet very complete. The intestinal lesions are probably due to the conjoined local influence of the bacilli and their toxines. To these influences may be added septic elements absorbed from the local inflammatory and necrotic areas.

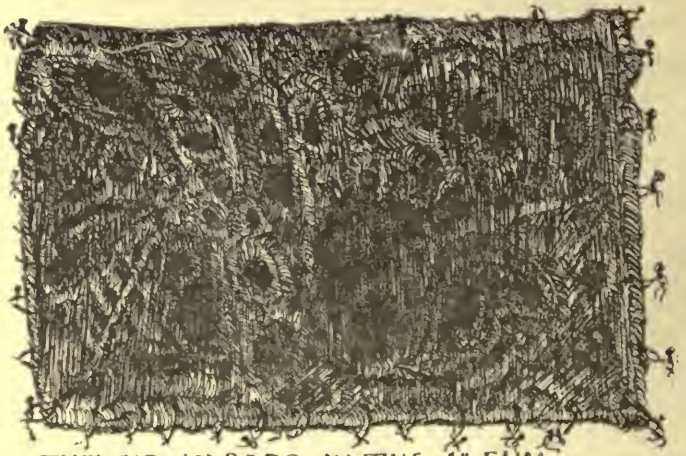
The characteristic *anatomical lesions* of typhoid fever are found in the alimentary canal, though it is important to recollect that the granular degeneration (cloudy swelling) of tissue, common to all protracted fevers, is marked in this disease, involving the muscular structure of the heart, this suggesting great caution during convalescence, lest the patient overtax his strength. According to Ziegler, the morbid changes in typhoid appear chiefly in the lower part of the ileum and the upper part of the colon; they are seldom met with much higher or lower in the intestine. The changes consist essentially of a necrotic inflammatory infiltration of the follicular structures and the parts around them, accompanied by a catarrhal inflammation of the rest of the mucous membrane.

"In the first few days of the attack the mucous membrane of the lower part of the ileum and its agminated glands of Peyer's patches are intensely congested and uniformly swollen. Soon the swelling of the patches becomes more marked, raised and winding ridges not unlike the cerebral convolutions in miniature appearing on their surface. The swelling extends more or less quickly over the whole of each patch, so that it has in general the look of a bed or garden plot projecting above the general surface. When the swelling is at its height, the ridges are generally leveled up, as it were, and are no more distinguishable. The surface of the patch is then smooth, or pitted minute depressions correspond to the sites of the individual follicles. The solitary follicles form rounded nodules by virtue of the same process.

"When this stage (of swelling) is complete, the patches and follicles, which at first were bright red in color, become pale and creamy white.

"The swelling of the patches and follicles is chiefly due to the extreme cellular infiltration of the mucosa and submucosa.

"The number of swollen patches varies much. Often but a small number or even a single one is markedly affected; while in other cases the affection extends upwards to the jejunum or downwards to the anus.



TYPHOID ULCERS IN THE ILEUM.

CICATRICES THE RESULT OF TYHOID ULCERATION.

"In the second week of the disease, partial disintegration and necrosis of the swollen patches usually sets in. The disintegration attacks the whole of the central part of the patch, or two or more parts of it simultaneously. The surface quickly assumes a frayed or

ragged appearance, and becomes yellow or brown from the action of the bile. Gradually the disintegrated tissue or slough becomes loosened at its base and edges from the surviving structures, and in a few days is cast off. After the separation of the sloughs, an erosion or typhoid ulcer is left, the floor of which generally looks smooth and clean. The borders of the ulcer at this stage are still swollen and infiltrated.

"The ulcers usually remain coextensive with or very slightly overpass the area of the infiltrated patches and follicles; they rarely invade the tissues beyond. Cases however occur in which, especially around the ileo-cæcal valve, extensive tracts of mucous membrane are attacked and disintegrated by the advance of the ulcerative process. In the vertical direction it seldom goes beyond the mucosa and submucosa. It is only when the infiltration of the muscular coat has been extreme that they too break down and ulcerate. In exceptionally severe inflammation the serous coat also may be attacked, but never to the same extent as the overlying layers; perforation and fatal peritonitis may occur in such a case.

"The processes of absorption and repair begin at various stages of the disease. If no necrosis takes place, the swelling of the patches goes down as the infiltrated material is absorbed; the patches thereupon become less stiffly turgid, and once more hyperæmic. Red corpuscles escape from the damaged vessels, and the tissue takes on a red or blood-stained tint which presently turns to a slaty gray. The infiltrated borders of the ulcers become reduced and softened, and hyperæmic by the same steps. Often enough considerable hemorrhage ensues, leading not only to hemorrhagic infiltration of the tissue but to actual escape of blood into the intestinal canal. As the healing process goes on, the softened and overhanging borders of the ulcer become adherent to the floor; the latter is gradually covered over with delicate granulations, and soon receives an investment of epithelial cells."

Fortunately, the site of a typhoid ulcer heals without contraction, and intestinal stricture does not follow the extensive ulceration that so frequently attends ordinary suppurative processes. A smooth, shallow depression remains for a long time, presenting a slaty-gray color, and devoid of glands and follicles.

The mesenteric glands are more or less involved, those whose absorbents correspond to the portions of the intestine principally affected showing the most important changes. They become enlarged at the outset, and after the tenth or fourteenth day begin to soften, their contents becoming friable at first, and later degenerating into a pus-like fluid, mixed with sloughs. Finally, the glands become

tough, contracted and shriveled. During the period of softening they may burst into the peritoneum.

The *spleen* becomes congested and softened, its cells undergoing granular degeneration. The *liver* partakes of similar characteristics, and the gall-bladder may be the seat of catarrhal or diphtheritic inflammation. During the late stage of the disease the bile may be watery in consistence, colorless, and acid in reaction. *Peritonitis* may occur from extension of the intestinal irritation; from intestinal perforation; from rupture of mesenteric glands, or spleen; or from perforation of an ulcer in the gall-bladder. The *kidneys* may become congested, and the tubules choked up with detached epithelium, owing to granular degeneration of the cellular elements. The congestion may also extend to the mucous coat of the *bladder*. *Pulmonary* congestion is almost always found among the post-mortem evidences of the disease. The *blood* is dark and fluid, and the white corpuscles are increased in number. Disintegration of the red corpuscles is also noticeable at times. No particular changes are observable in the *nervous system*, though there may be an excess of serum in connection with the brain and its membranes.

Symptoms.—INCUBATION.—This lasts from two to three weeks. During this time there are no peculiar symptoms, and in many cases the patient does not suspect that anything is wrong. In other cases there may be a feeling of prostration with headache, vague pains, loss of appetite, and sleeplessness.

INVASION.—The onset of typhoid fever is often so gradual and insidious that the actual time of beginning is not appreciable to the patient. Indeed, in some cases he may not be indisposed enough to give up business and go to bed before the fifth or sixth day. There is not the marked chill which ushers in many forms of fever, and though there may be chilly sensations in the start, these do not usually amount to a pronounced rigor. *Headache* is a more constant and urgent symptom, the pain usually invading the frontal region at first, though as time passes it finally becomes more general. Attending this there are giddiness, roaring noises in the ears, lassitude, fugitive pains, restlessness and insomnia, loss of appetite, furred tongue, nausea, epistaxis, and usually diarrhœa, with abdominal pain. Attention to the state of the temperature, during this time, will disclose the fact, usually, that it is gradually rising, each succeeding day marking an advance upon the preceding one of less than a degree, and each morning showing a remission of about one degree below that of the previous evening. This gradual accession continues for six or eight days, by which time the maximum is reached, and the increased pulse-rate and arrest of secretion having been keeping in

touch with the rise in temperature, a high grade of pyrexia has finally been established.

The *pulse* is now (beginning of second week) running from 100 to 110 per minute in the evening, with slightly lessened rate in the morning during the remission, the temperature ranging as high as 105°–109° F. The *skin* is hot, dry, and husky, and there is marked restlessness, the patient complaining bitterly of headache, or else being delirious (delirium first appearing during the night to pass off during the following day, but soon becoming continuous), with nocturnal aggravation; the tongue has taken on evidence of encroaching depravity of the blood, being coated white with reddened tip and edges or loaded with foul accumulations, while there may be nausea and vomiting, even during the first week.

The *fastigium* is now reached, and we find the patient loses flesh and strength rapidly. The pulse becomes dicrotic and feeble, as the heart loses power; the tongue becomes shriveled, dry, and brown. Active delirium exists, which passes into a condition of low, muttering semi-coma in the *third week*. Now occur stupor, subsultus, tremors, involuntary evacuations, and other evidences of profound exhaustion and prostration. Sordes collect upon the sides of the mouth and teeth in sufficient quantity to form crusts, and become more abundant as the disease progresses. The countenance presents a pale, leaden appearance, with a hectic flush in the center; the face lacks expression, the patient sleeping with the mouth open, and tending to slide downward, toward the foot of the bed.

The morning remissions become lengthened during the third week, and as these increase in length and the maximum temperature diminishes, the morning decline finally reaches the normal during the *fourth week*, the pulse gradually becoming stronger and less frequent, the cerebral symptoms disappearing, and delirium giving way to restful slumber, the tongue becoming cleaned and moistened, and the skin relaxed and softened. During the waking periods the patient now (fifth week) complains only of prostration and hunger, his appetite and weakened mental condition conspiring to render him peevish and exacting, in matters pertaining to a proper diet.

As early as the first week—by the sixth day—there will be found pain and tenderness in the right iliac fossa. Slight pressure elicits pain now, attended with gurgling, and the sensitiveness becomes more marked as the disease progresses. Examinations of this character should be made with the palm of the hand, and the pressure be gentle. By the beginning of the second week *tympanites* begins to appear, the abdomen gradually becoming distended and drum-like. This usually becomes extreme as the disease advances, being

due to a collection of gas in the large intestine. The tympanites is an indication of serious pathological change in the alimentary canal, and so long as it continues it is patent evidence that the patient is in a precarious condition.

A common though not constant symptom of typhoid fever is *diarrhœa.* This may be present during the first week, but may not appear until the third, though it is liable to be most prominent during the second. The evacuations are peculiar, being of a greenish-yellow color, and being described as "pea-soup discharges." Sometimes they are dark in color, resembling coffee-grounds. They are alkaline in reaction, and upon standing deposit a granular sediment, the upper portion being watery in character. In mild typhoids, as well as in some severe ones, diarrhœa may be absent.

Intestinal hemorrhage is said to occur once in about every twenty cases. It is the result of sloughing of an artery in the intestinal wall, and is a very dangerous complication. The hemorrhage may occur without any external evidence of the discharge, rapid fall of temperature and great prostration attending. In other cases, the blood flows in large quantity from the bowel. Slight hemorrhages from the mucous membrane of the bowel may occur early in the disease, but they are of trivial consequence, as they consist merely of capillary oozing, similar to that of the early epistaxis. Arterial hemorrhage rarely occurs earlier than the latter part of the second week, and is more likely to occur during the third. If the intestinal structures be fortified during the preceding time, there is diminished risk that this accident will occur. The treatment from the beginning should look toward a favoring of the integrity of the intestinal structures.

Between the seventh and twelfth days the characteristic *eruption* appears. It is found most abundant upon the abdomen and chest, but may be isolated over other parts of the body. It appears in minute, round, rose-colored spots, slightly elevated above the general surface, disappearing upon pressure, but returning immediately afterward. Each spot remains visible for three days, but successive crops may appear during a period of ten or twelve days. In some cases they are so faintly marked that great care may be necessary in order to detect them. They possess little significance therapeutically, but are considered a diagnostic symptom of true typhoid fever, by many. They are, however, sometimes absent.

During the first two weeks, the *urine* is scanty and dark colored, and shows a high specific gravity; after the second week the quantity is increased, and during convalescence it becomes pale and abundant, and its specific gravity is lowered. It is asserted that upon an aver-

age the amount of urine voided during typhoid fever is greater than the normal amount during the same length of time; but this is probably due to the fact that the diet is largely liquid now, thus providing for a greater amount of fluids in the body. Albumin is occasionally found in the urine of typhoid patients, though not in many instances.

The *nervous* symptoms of typhoid fever are prominent throughout its course, in most instances. The mental symptoms during the early part of the disease are often those of apathy, the patient being torpid, careless, and unimpressible, answering questions slowly, and giving little heed to his surroundings. In other cases, however, extreme restlessness may be manifested in the start, the patient changing his position frequently, tossing about almost constantly, and failing to find rest anywhere. *Active delirium* often sets in with the establishment of the fastigium, it sometimes being necessary to employ force to prevent the patient from doing himself or his attendants harm. At other times, though this condition is liable to come on later, there is more of a subdued delirium, characterized by dreamy aberrations attended by incoherent mutterings—*typhomania*. This condition is pretty well established by the latter part of the third week or fore part of the fourth. Gradually, in favorable cases, this passes into a somnolent condition, and the fever runs its last few days and terminates with the patient sleeping most of the time.

The *special senses* are often involved. Impairment of hearing is a common symptom, it being necessary to speak above the ordinary tone to elicit an answer. Vision is also impaired, for a time, in many cases. Paralysis of the sphincters is a prominent symptom in some instances, the evacuations passing involuntarily, and necessitating the use of diapers to protect the bedding, while in other cases retention of urine from loss of vesical power may demand the regular use of the catheter.

Temperature.—The temperature of typhoid fever—the typical temperature—shows a gradual rise of a little less than a degree per day above the maximum of the preceding one, with a morning remission of one degree for the first week or about eight days. During the second week the maximum of each day remains about the same as that marked on the eighth day, with a morning remission of near one degree. During the third week the remissions become more marked, though the maximum still remains at about the same height. During the fourth week the remissions become still more marked, while the maximum declines day by day, until the normal line is reached during the decline, actual intermissions finally occurring.

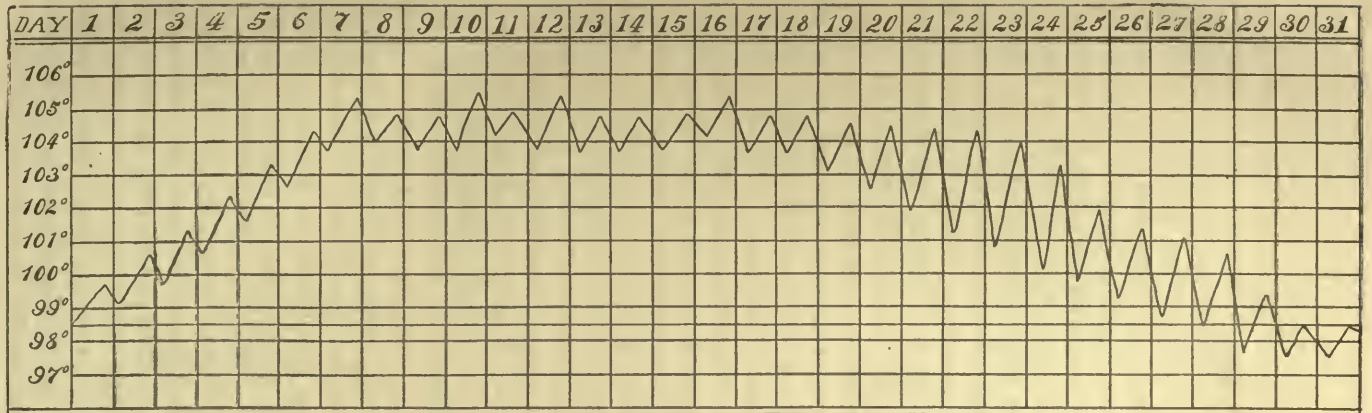
During the last two or three days of decline, the periods corresponding to the morning remissions may be marked by slight subnormal temperature, and this may continue into convalescence.

Usually the temperature begins to rise about the middle of the day on the first day of the fever, and the exacerbation continues until six or eight o'clock in the evening, when it remains stationary until about midnight; then it begins to decline and continues to do so until six or eight o'clock the following morning, when the minimum of the remission is reached for that day; the temperature now remains about at this point until the middle of the day, when it again begins to rise, and remains elevated until midnight, as before, when it again begins to decline, continuing to do so until six or eight o'clock in the morning, when it remains about stationary until the following noontime, when it again rises as before. This rotation of rise and decline—the temperature rising a degree higher than the maximum of the preceding day and falling about a degree each intermission—thus continues for the first week.

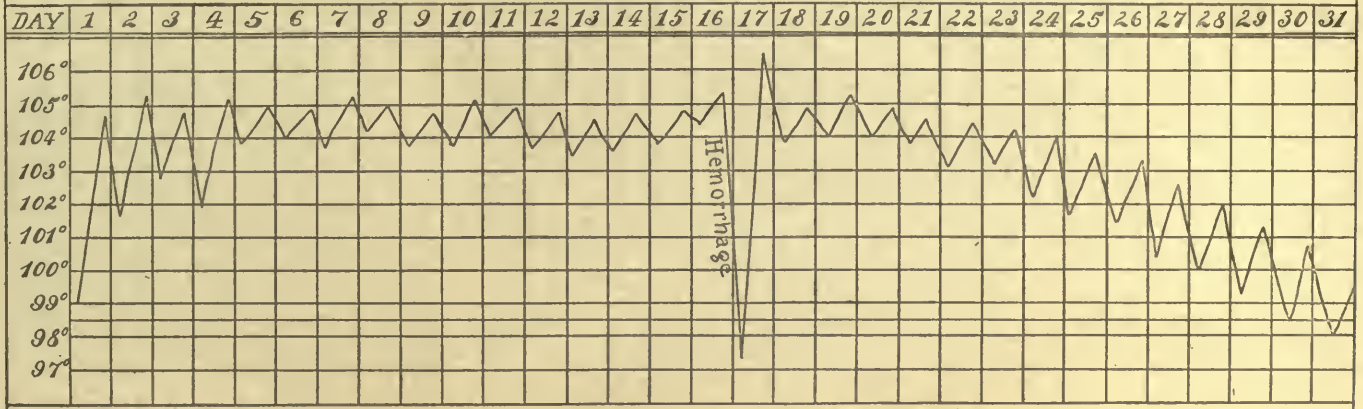
While such is the usual course of the disease, *marked variations* may occur as the result of complications, and these variations may often be considered as the most positive index of such complications. Treatment may modify the temperature very much, and some drugs, as antifebrin, possess the power of lowering the temperature rapidly, though not always safely. Relapses are not uncommon in typhoid fever, and are marked by gradual rise of temperature. Intestinal hemorrhage, when profuse, is usually marked by a sudden fall of temperature, to the extent of two or three degrees. When this fall occurs during the second or third week, and is accompanied by marked prostration, it is pretty good evidence that an artery has been opened in some intestinal ulcer, and that profuse hemorrhage has occurred, even though no blood has been voided in the dejections. Perforation of the bowel is soon followed by a rapid decline of temperature, which is succeeded by abrupt and extreme elevation, and collapse.

Occasionally, the stage of invasion is abruptly announced by a *sharp chill*, which is followed by a high temperature without the gradual rise, the case opening at once into the fastigium. When malarial complication exists, the temperature may resemble that of intermittent fever until the abdominal lesions develop, there being a daily chill, followed by fever and sweating, for the first week.

Cases in which the temperature declines to normal at the close of the second week are spoken of as "aborted typhoids," the early convalescence being due to the fact that the intestinal lesions subside without going on to ulceration.



TYPICAL TEMPERATURE OF TYPHOID FEVER.



ATYPICAL CASE WITH HEMORRHAGE OF BOWEL.

Relapses and *recrudescences* are not to be overlooked. Relapses are due to reinfection of the intestine from sloughs which have been cast off from above, in cases which have not received proper medication during the first attack. They are not common, but are said to occur in from 3 to 18 per cent of all cases, though exceedingly rare in Eclectic practice. If they occur during the fastigium, there is merely a prolongation of the stage of active fever. Most frequently they occur from five to eight days after the termination of the primary attack, though this period may be prolonged to twenty-five days. When they thus occur, the temperature curve is repeated, though it is very much shortened, the stage of invasion ending in from three to five days, and the other stages accordingly, ten to fourteen days being occupied in this second course. Recrudescences are temporary rises of temperature occurring during convalescence, from dietary indiscretions or overexertion. These occur suddenly, and, in favorable cases, terminate in from one to five days.

During convalescence, the temperature may remain persistently elevated two or three degrees for several weeks, this being due to unhealed ulcers in the intestine, which only recover by slow stages.

Diagnosis.—The regular and gradual rise in temperature and morning remission during the first week, the abdominal tenderness and intestinal irritation, the rash, and the infectious character of the disease, will be sufficient to warrant the diagnosis. It might be supposed that as this disease is dependent upon the presence of the typhoid bacillus in the alimentary canal, a microscopical examination of the dejections would readily settle the question in any doubtful case. But it seems that there are many difficulties in the way of detecting these bacteria here, and that it is only in a small proportion of the cases that they can positively be identified. Dr. Adolph Gehrmann, an authority upon bacteriolgy, remarks (Fort Wayne *Medical Magazine*):

"Reported demonstrations of the isolation of typhoid bacillus from the evacuations of patients are not numerous. Pfeiffer, Karlinski, Frænkel and Simons have so reported. In the case of the last-named experimenters, out of eleven separate attempts by means of direct cultivation by Koch's plate method, success was attained in but three instances. The diagnosis here was made by obtaining cultures of typhoid bacillus by selecting colonies on the plate cultures peculiar to that organism. A method of this kind is most difficult, as only the smallest quantity of discharges can be taken, and the dilution must be extreme, in order to separate them widely enough to obtain isolated colonies. Where bacteria of all kinds are in such great numbers, there must first be some quick means of separating

the organism in question from all others, and lastly some means of establishing its identity. The separation of typhoid bacillus from the majority of its associated bacteria is not a matter of great difficulty, but its final identification has proven a most serious obstacle. Typhoid bacillus is not a distinctive species. Its similarity to *bacillus coli communis* is so close that the proposed distinctive features have been overthrown, one after another. Apparently there is a group of bacteria having typhoid bacillus at one extreme and bacillus coli at the other, while between them is a gradually changing series of varieties. Much more time has been devoted to the study of these peculiarities and to the presence of the group in water supplies, than to the relation of these bacteria to the intestinal canal. This variability of typhoid bacillus has always occasioned the greatest difficulty in studying the biology of the organism itself, or in investigating the cause and nature of the disease."

Prognosis.—Though a disease of grave aspect, the mortality, under proper treatment, ought not to be great. In ordinary epidemics, 5 or 6 per cent ought to be a large death rate, where modern Eclectic methods of treatment are pursued. There is a marked difference in the severity of different epidemics, the abdominal symptoms being exceptionally severe in some, while in others the cerebral symptoms are aggravated. Hospital reports usually show a larger percentage of deaths than commonly occur in the private practice of Eclectic physicians, and the custom of recommending such patients to the care of hospitals is not to be commended. During a recent epidemic of typhoid fever in this city (Oakland), the mortality in Fabiola Hospital (Homeopathic) was remarkably great, though not many deaths occurred in private practice, and these almost entirely under old school treatment.

Hyperpyrexia, aggravated delirium, hemorrhage, and peritonitis, are unfavorable symptoms, and should lead to a guarded prognosis.

Treatment.—The treatment of typhoid fever may be divided into *preventive* and *restorative*. At the commencement of an epidemic, the conscientious physician will make early inquiry into the origin of the disease, and see that as few persons as possible are exposed to the infection. In rural districts, the water supply is usually the source of contamination, the disease having previously been communicated to some well or spring from which the family or families affected are using, all who drink the water receiving the germs into their systems. In cities, such water may be used to dilute the milk distributed before it is conveyed from the country, or in washing the cans in which it is transported, and the disease thus be distributed. In such cases, the health officer who does his duty will see that the

supply of milk containing the germs of the disease is ferreted out, and shut off by strict quarantine. Wells or springs suspected or known to be contaminated, should be avoided, and where there is positive evidence that such a place has been vitiated, it should be filled up and a new one dug. Wells or springs in the neighborhood of privies, barn-yards or sewers, should be looked upon with suspicion, and the water avoided.

As the specific poison exists in the evacuations of typhoid patients, great care is demanded during the course of the disease that these be so treated that there will be no possibility of contamination resulting from them. A porcelain bedpan should receive the discharges, and before use this should be charged with half a pint of saturated solution of sulphate of iron or copper. A more convenient plan, considering the preservation of the bed, is to sprinkle the bottom of the vessel over with dry powdered sulphate of copper, while immediately after the evacuation crude muriatic acid or a strong solution of copperas is added. It should now be allowed to stand a sufficient length of time for the chemicals to react thoroughly upon the morbid material. The disposal of the contents of the pan is an important consideration. When possible to do so, it should be emptied into a trench and immediately covered, new trenches being dug frequently. Care should be observed to see that the fecal material is not emptied upon the surface of the ground, nor deposited near the well or spring from which drinking water is obtained. If it becomes necessary to empty the evacuations into a closet-trap, this should be frequently scalded with boiling water, and none should be emptied here until thoroughly disinfected.

Care should be taken regarding the disposal of cloths, wearing apparel, and bedding, which may be soiled by the discharges, that no contamination is conveyed to the non-infected. Cloths of no particular value may be burned, and other fabrics should be immediately washed, being thoroughly boiled during the process of cleansing. A very good plan would be to immerse the clothing in a 1–1000 solution of bichloride of mercury before any attempt at cleansing was made, the immersion to be continued an hour. Nurses should be careful about communicating the infection with their hands.

The rooms occupied by typhoid fever patients should be free from opportunity for the breeding of fomites. Carpets and tapestry would be best dispensed with, as well as all furniture, except such as is needed for patient and nurse. There should be a free supply of pure air, though the temperature should not be far below 60° F. The room should be kept quiet, and all causes of disturbance to the patient avoided.

In order to avoid any chance of injury to the bowel, the diet should be liquid in character, rice water and malted milk or Mellin's food constituting the principal part, all solid food being dispensed with; and fruits should not be allowed under any consideration. The food should be given at regular intervals, as promptly and regularly as medicine, and in small quantities, repeated as often as every two or three hours.

Owing to the marked debility of the tissues brought about during this disease, and the tendency to hypostatic congestion resulting, the patient should not be allowed to remain for any considerable length of time in one position. As he is liable to soon become helpless and unconscious, the nurse should be instructed to turn him from side to side and from side to back frequently, especially during the day, and this precaution should be so observed that he shall not remain in one position more than four hours at a time, unless intestinal hemorrhage or other complication render it inadvisable to disturb him. In all manipulation, the delicate condition of the bowels should be recollected, that injury be not added to that inflicted by the pathological changes which are going on.

Good nursing shows a better record of mortality in the management of this disease than that of old and harsh methods of treatment. Cathartics should be carefully avoided. Opiates and mercurials are dangerous in the extreme. Quinine and other bitter tonics, as well as stimulants, should only come in late in the course of the disease or during convalescence, if at all.

What are the rational *therapeutic indications* in the treatment of this affection? Where do we find the most marked evidence of morbid action? Evidently, the severity of the affection is manifested upon the intestinal structures, and the case becomes grave in proportion as the pathological changes here are marked. It is true that there may be a cerebral complication in isolated cases which will render the prognosis serious where the abdominal symptoms are not severe, but this is hardly the rule, and is a rare exception. If we will direct the excellent means at our command to the relief of intestinal irritation, and to the fortifying of this part against pathological changes from the very beginning, we shall not find many cases to present very grave features.

There are two remedies which I think we may employ for this purpose with a great amount of confidence, viz., *baptisia* and *echinacea*. It is not necessary that we discriminate particularly as to "specific indications" in differentiating between the use of these agents here. Both possess a relation of a restorative character to sloughing mucous membranes, and a restorative character where necrotic conditions

are present or threatened, and both are recognized as valuable antiseptics. Echinacea is a remedy of especially valuable properties where a sedative, antiseptic, restorative, and vital stimulant are required in the one agent, and its excellent effects where meningeal irritation is present is an additional recommendation. I believe that most cases of typhoid fever may be safely trusted to the action of this remedy alone.

As there will probably be a demand for other treatment, we will not obstruct the opportunity for further medication by alternating these, but will administer them in combination. To half a tumbler of water add a drachm of baptisia and two or three drachms of echinacea, and order a teaspoonful every two hours. We will let this combination be the basic remedy, and will continue it until convalescence has been announced by a normal temperature. It would not usually be a pleasant combination, as to taste, but the typhoid patient will make no objection to it, as his sense of taste has probably been abolished, for the time.

With this treatment directed toward the prominent pathological lesion, we will hold our reserves in readiness for such complications as may arise.

Hyperpyrexia.—A prominent and serious symptom of many cases of typhoid fever is an excessively high temperature. Rapid destruction of tissue must result in such cases, and danger to tissues naturally jeopardized, such as the intestinal walls and brain, is very much increased. When the maximum temperature is above 106° F. during the stadium, the condition may be considered hyperpyretic. There are various opinions among physicians of our faith respecting the proper treatment of this condition. Professor Scudder taught, and he has many followers who agree with him, that the febrile feature of typhoid fever is successfully met by the use of the special sedatives—aconite, veratrum, and gelsemium. The argument is that these remedies, properly adapted, administered in minute doses, and frequently repeated, control and strengthen the action of the heart and bloodvessels, lessen excitement, and promote secretion, thus lowering the maximum of the temperature throughout the course of the fever. I have been a believer in this doctrine myself, and I am satisfied that the plan, if not overdone, is followed, in a large percentage of the cases, with successful results. Where this plan is to be pursued, the proper remedy should be selected upon certain well-known principles (see author's "Principles of Medicine"), and administered in minute doses at frequent intervals throughout the course of the disease.

However, while I am a firm believer in the special sedative treat-

ment of fevers generally, my recent experience has led me to believe that little real benefit follows the practice in the management of this form Typhoid fever is a mild form of septicæmia, and seems unimpressible by such means, when compared with results in other forms. The thermometer will not manifest any decided impression from this plan of treatment in the majority of cases. Still, there is no gainsaying the fact that these remedies may improve the patient's chances, by soothing nervous excitement and promoting rest—shortening the period of active delirium.

What I consider a more pronounced antipyretic in such cases as are here under consideration (hyperpyretic), is the *salicylate of ammonium*. The formula is as follows, though the salt may now be obtained, prepared, in the drug market:

℞ Salicylic acid, ʒii; carbonate of ammonium, ʒiii; aquæ menth. piper., fʒiv. M. Dose, a teaspoonful.

This may be administered every two hours, in alternation with the combination of baptisia and echinacea, until the temperature has fallen below that of hyperpyrexia. Indeed, this remedy may be continued throughout the course of the disease with gratifying effect, in many instances.

Another excellent means in hyperpyrexia, and one which may be used in conjunction with this or employed independently, is the cold *abdominal pack*. This may envelop the entire form, but one large enough to reach from the axillæ to the thighs will usually afford better satisfaction. The bed is protected with an oilcloth or rubber blanket, and half a sheet is wrung out of cold water and wrapped about the body as already suggested, to be renewed every two hours, until no longer needed. A good substitute for this application is a large clyster of cold water, though here the patient must aid in retaining it, a condition requiring possession of the mental faculties, and one not always available. In making use of rectal injections here, the weakened condition of the bowel must be borne in mind, and the application of much force carefully avoided.

The administration of large doses of *quinine* for the purpose of lowering the temperature in these cases has been strongly advocated by certain old school authors, and some reputable writers of our own school have indorsed the doctrine. My experience has been that such medication usually aggravates cerebral symptoms, and does not markedly lower the temperature. However, circumstances alter cases, and it may be possible that certain epidemics or certain localities may present us with cases where such treatment would be strictly the proper thing. However, I would enjoin caution in this method until the fact was proven clinically in at least one case of an

epidemic, before beginning the indiscriminate use of quinine as an antipyretic in typhoid. As a general rule, the proposition is bad.

There are those who extol *acetanilide* as an excellent remedy for this purpose. It is asserted that an immediate fall of temperature follows the administration of from three to five grains, and that a period of two or three days ensues before the temperature rises to a point demanding a repetition of the dose. But it is to be remembered that this remedy is markedly depressing, and upon theory, not a desirable agent to administer in such a condition of prostration as is found in this disease. It would be fair to expect a large mortality to attend such treatment, even though we be assured that such is not the case. I would be inclined to regard this remedy with grave suspicion, however, until fully convinced by observation of some one else's practice that the vaunted benefits can positively be derived with safety.

Delirium.—The delirium of typhoid fever is sometimes appalling. The patient may be so furious as to seemingly force the responsibility upon the physician of attempting to control the ravings with drugs. As this goes on day after day, attendants or friends may urgently ask that strong drugs be used to promote slumber. In such cases the physician must preserve the greatest moderation as to the character of his medication, seeing that active narcotics are strictly avoided, as there could hardly be a more dangerous place for their exhibition. It may be true that attendants become worn out in their efforts to prevent the patient from leaving his bed, and it may seem that the efforts of the patient himself may end in fatal exhaustion, but there is much less danger of this than of the effects of opiates. Minute doses of aconite and rhus tox. may afford some benefit, or small doses of belladonna, 3x dilution. Possibly, though not probably, small doses of bromides may benefit. Passiflora is not of much use as a calmative during febrile action. Sulfonal may benefit some, but is not likely to. Cold cloths to the head, and sometimes ice-bags, may afford good results. The general condition and benefit of the patient should, however, always be held paramount to that of special means for the relief of what is but one of the phases which this fever almost always presents. In due time the active delirium gives way to that dreamy wandering consequent upon the exhausting effects of the first onset, and the better the management has been up to this time the better the patient's chances will be to survive the further ordeal. Sometimes, where there are presented quite vividly the indications for gelsemium—flushed face, bright eyes with contracted pupils, full, bounding pulse, etc., the zealous Eclectic (or specific medicationist) may be tempted to push this

drug beyond safe bounds, and cause debility of the circulation, from which the patient may rally with difficulty. It is well to remember that recovery from this fever is a sort of evolution, through which the proper treatment consists in safely guiding the case to a successful issue by fostering the processes of life so far as possible, and avoiding all measures which might interfere with the best performance of these functions.

The beneficial effects of tepid baths in such cases should not be forgotten. The restlessness and furor of the stage of active delirium should be met with frequent sponging, and the nurse should possess the requisite knowledge to prompt persistent resort to this measure. Sponge baths should be applied several times a day, and it will soon be noticed that the patient rests better, for a time, after this application.

Gastric Complications.—The stomach is frequently disturbed by morbid conditions which interfere with the action of remedies. Gastric irritation may be present, marked by nausea, rejection of food and medicine, and restlessness; the tongue will be pointed, and reddened at the tip. Here we will usually be able to correct the condition with small doses of aconite and rhus tox., and it may seem best to dispense with all other treatment for a day or two, until the stomach has become well settled. Add ten or fifteen drops of rhus tox. and five drops of aconite to half a glass (four ounces) of water, and order a teaspoonful every hour. This may possibly fail after a fair trial, but is hardly likely to; however, two grains of subnitrate of bismuth every two hours may then be tried, and, in event of failure with this, minute doses of ipecac or peach bark infusion.

Another gastric complication which is very common in the course of typhoid, is excessive acidity of the stomach. This is marked by the broad, flabby tongue, evenly and thickly coated with a *pasty white* coating. As sepsis is more or less marked there will be an element of color in this coating, it often being described as "dirty." Sodium sulphite is here the corrective, and this agent should be administered in one- or two-grain capsules, every two or four hours until the tongue cleans or presents a different aspect. It is important to correct such a condition in order that other remedies may be readily appropriated.

Special Septic Conditions.—While typhoid fever is of itself a markedly septic disease, and while the use of echinacea and baptisia has been advised throughout, partly for their antiseptic influence, there are special conditions liable to arise which may demand other remedies of this class, though not likely to be marked if these remedies are continued from the beginning. Sulphurous acid is an

agent which is sometimes urgently demanded in the treatment of this disease. The condition which requires it is indicated by *brown coating* on the tongue and sordes on the lips and teeth; here sulphurous acid should be given in twenty- or thirty-drop doses, well diluted, every two or three hours. The *beefsteak* tongue—clean, dark-red, slick—may appear toward time of convalescence, and will demand the use of mild acids, such as acid drinks or dilute muriatic acid. Of the latter, ten to twenty drops may be given every four hours, until the characteristic condition of the tongue has given way to a natural appearance.

Diarrhœa.—This symptom is a very common one in typhoid, and one which it might seem necessary to control. However, it is to be recollected that it is but a result of the catarrhal inflammation of the mucous membrane of the lower bowel, and the rational management will consist in the use of means which will control the intestinal irritation, to which we have already directed echinacea and baptisia. There would be no logic in attempting to control this difficulty with astringents; and it would only be a return to the crude practice of obsolete medicine. Salol has been highly recommended in certain quarters, on account of its antiseptic influence in intestinal sepsis. But the advocates of this remedy are those who are not acquainted with Eclectic remedies, such as echinacea and baptisia.

Aside from the use of proper antiseptics and correctives from the beginning, the diarrhœa may properly be allowed to take its course, as general treatment will serve a better purpose than local measures. If any particular remedy were to be recommended, it would be a decoction of erigeron canadense plant, a remedy that is readily obtainable in the autumn season. This cannot do any harm, and may be drunk freely.

Tympanites.—Extreme distension of the abdomen may seem to demand special attention. With the improved treatment I have called attention to, there will be but few cases where it will be prominent; however, an occasional case may demand attention. An old and useless (as it seems to me) practice consists of the application of turpentine stupes to the abdomen. The room and surroundings are thus filled with the disgusting fumes of turpentine, that everybody in the vicinity may recognize the fact that something is being done. This may afford some satisfaction, but the utility of the measure is doubtful. Rectal injections of clysters containing asafœtida also have their advocates, and it is possible that there may be more benefit derived from them. Intestinal antiseptics, administered internally, as salol or naphthol, are more to be commended.

Intestinal Hemorrhage.—The slight hemorrhages of capillary

origin, and which occur early in the course of the disease, require no treatment; but after the second week intestinal changes may have occurred, which will render hemorrhage liable to escape from the arteries which supply the intestinal walls, and which will demand prompt arrest, when this is possible. It does not seem that there will be much danger of hemorrhage when the treatment here suggested is faithfully followed throughout the early part of the disease, though it is true that some epidemics are attended by more severe intestinal lesion than others, and that the condition of the patient prior to the attack may predispose him to deep necrotic changes here; but I have found little reason to expect intestinal hemorrhage of serious nature, in my experience.

A patient with intestinal hemorrhage should be kept strictly quiet, and should have decoction of erigeron canadense administered, two-ounce doses every half hour, until the active hemorrhage is arrested. If oozing continues, the remedy may be repeated at longer intervals, until the discharge is entirely arrested. It has been advised to apply ice-bags to the abdomen and administer hypodermic injections of ergotine. A pill containing acetate of lead, gr. ii, and extract of opium, gr. x, administered every four hours, has been known to succeed where there was persistent oozing.

The collapse which follows intestinal hemorrhage may require hypodermic injections of strychnia, these being employed in fifteenth or thirtieth of a grain doses, and repeated every two hours until reaction takes place.

Constipation.—In a few cases constipation may be present, and pressure may be brought to bear to induce the physician to administer a cathartic. It would be bad practice, however, to administer opening medicines to any one affected with enteric fever. It would be better to allow the bowels to remain ten days without an evacuation than to commit the error of administering a cathartic. True, there might be some urgent symptom requiring a violation of this rule, but the danger of allowing ample time for nature to regulate this condition is not usually comparable with that of forcing an evacuation with cathartics. Mellin's food is an excellent article of diet where constipation is present, though the condition will hardly arise unless pure milk is employed as food, which should not be allowed in any case.

Convalescence is a critical period in the management of typhoid fever, as the patient is prone to indulge in exercise and diet which may prove fatal in their results. Fatal peritonitis may follow the early ingestion of solid food, and the patient should be solemnly warned of the danger incurred by too early indulgence in such

matters. During this period the *diet* should be restricted to milk, cream, gruels, jellies, and animal broths. Ice-cream, in moderate quantities, may be permitted, but solid food, such as meats, vegetables, and fruits, should be strictly forbidden.

As healing of the intestinal ulcers is not completed until two or three weeks of convalescence have passed, the patient should remain in the recumbent position, part of the time at least, and avoid all exercise, except walking about the sick-room, during that time. The use of solid food should be begun with very small quantities at a time.

Supplemental Therapeutics.—A few other means employed in the treatment of this disease, are worthy of mention.

Intestinal Antisepsis.—Modern old school authorities regard this with great favor, some asserting that through it the course of the disease may be aborted. The principal remedies used are salol, beta-naphthol, salicylate of bismuth, creosote, iodide of potassium, and some other antiseptic agents.

Further suggestions on the treatment of this disease may be found in "Dynamical Therapeutics."

II. TYPHUS FEVER.

Synonyms.—Ship Fever; Jail Fever; Irish Ague.

Definition.—This is an acute, contagious disease, characterized by sudden and marked prostration; abrupt invasion of fever, with rapid rise of temperature; a peculiar rash; marked nervous symptoms; and a termination by crisis, about fourteen days from commencement.

Etiology.—The poison of this disease has not yet been identified, but it is probably similar to that of other infectious diseases, viz., a microscopic germ, capable of producing the disease by rapid multiplication and the generation of ptomaines in a healthy person, after entering the system, upon exposure to the infection. Near approach to one affected is usually a requisite to infection, and the carrying of the disease in clothing, as in small-pox, is of very rare occurrence. It rarely travels from house to house, and is usually communicated to those in constant attendance, instead of to occasional visitors, nurses and house-physicians being much more likely to contract the disease than the visiting physician. Loomis, in his work, "Practical Medicine," relates that during an epidemic of typhus fever which prevailed in New York, from 1861 to 1864, of those who attended to washing and packing away the clothing of patients brought into the hospital, after it had been removed in the reception room, all, even to an individual, escaped the disease,

while every one whose duty it was to assist in carrying them from the reception room to the wards, took the fever.

It would seem, then, that near approach to those affected is essential to the contagion, and that this is modified much in open air, our author stating that less than two and a half feet measure the average limit of infective distance from an affected person, under such circumstances.

While Loomis states that the disease seems not readily propagated by fomites alone, most authorities assert that it can be so conveyed. The hospital experience referred to in former paragraphs, however, appears to throw much doubt on the statement.

Only the great seaports afford cases of this kind on the American continent, and these are usually brought there by vessels entering from foreign parts. Europe seems to be the geographical center of origin, the disease being common in Russia, England, and Ireland.

The disease is most liable to occur among those who are occupying crowded quarters, such as old tenement-houses, jails, and other illy ventilated public places. Owing to improved sanitary conditions, typhus fever occurs but rarely, in modern times.

A single attack affords an individual immunity against subsequent ones.

Pathology.—The pathological lesions of the tissues and blood found in typhus fever resemble those of typhoid, in many particulars. The *blood* in typhus is found darker in color than normal, and when abstracted during life it is seen to have lost its normal property of coagulation; and, if a clot forms, it is brittle and pultaceous, the mass seeming to be devoid of fibrin elements. The red corpuscles are increased in number at first, but they diminish as the disease progresses; there is also a change in the salts, the blood rapidly undergoing ammoniacal decomposition when drawn from the body. Before standing, the blood contains urea and ammonia in excess. Microscopic examination shows many of the corpuscles degenerated, broken up, their edges irregular and serrated. The coloring matter thus set free stains the lining of the bloodvessels, heart, and other tissues.

Enlargement and friability of the *heart*, *lungs*, *liver*, and *kidneys* are not so marked usually as in typhoid, though the tendency to cloudy swelling and granular degeneration of the voluntary muscles, heart, kidneys, and other internal organs, is present. Pultaceous clots are often found in the cavities of the heart, or adhering to the walls of the larger bloodvessels. Splenization and hypostatic congestion of the *lungs*, as well as pulmonary œdema, are common results of this disease.

The *brain* differs much from that of typhoid fever in its post-mortem appearance. The vessels are here more or less congested, and the sinuses and large vessels are often engorged with blood, while the brain of the typhoid fever patient presents an anæmic appearance. Sometimes—and this may be characteristic of certain epidemics of typhus—there may be more or less extensive exudation of serum into the meshes of the *pia mater*, instead of marked congestion. Sometimes this effusion is turbid, suggesting meningitis as a complication. In these cases the *arachnoid* will be dotted over with yellow or yellowish-white spots, and its glistening appearance will be lost.

The *abdominal* lesions of typhus are not characteristic, and this will serve to distinguish it from typhoid, should any confusion exist. There may be congestion of the intestinal glands, with tendency to ulceration, but such a condition prevails more or less in scarlatina and measles, and is not distinctive of typhus. The marked and distinctive ulceration which characterizes typhoid is not present in typhus fever.

Glandular enlargements constitute a prominent feature of the pathology of typhus fever. The superficial cervical glands, and the parotid and sublingual, are often so much swollen as to interfere with deglutition. This marked swelling may sometimes apparently be the immediate cause of death.

The *inguinal* glands are also often swollen, so much as to retard the venous circulation from the lower extremities, and cause extensive swelling of these parts. Sometimes the irritation extends to the *veins*, and a condition resembling phlegmasia dolens results. Again, the *cellular tissues* may be involved and suppuration occur, resulting in large abscesses.

The *special senses* are not so markedly involved as in typhoid, and the *digestive organs* are not much disturbed, in the majority of cases, vomiting and diarrhœa being of comparatively rare occurrence.

Symptoms.—INCUBATION.—This may last from a few hours to two weeks. During this time there may be ill-defined sensations of discomfort, with dull headache, loss of appetite, fugitive pains, and other premonitory symptoms; but such indications are usually absent, the onset being unannounced.

INVASION.—The stage of invasion is abrupt. Premonitory symptoms, such as malaise, headache, insomnia with restlessness at night, nausea, anorexia, etc., may mark a few days of the latter portion of the period of incubation, but often the first symptom is a decided chill, which is short, sharp, and sudden; this is followed by fever, with rapid rise of temperature. Sometimes the chill is not marked, and only slight chilly sensations announce the onset. Following

the chill is a marked headache, which steadily increases in severity. It involves the frontal region, and soon becomes intense. Severe pains in the back and limbs attend, and as febrile symptoms, with a rapidly rising temperature, come on, a sense of *extreme prostration* overpowers the patient. Loomis relates that at one time, while he was making his visits in a typhus fever ward, his house physician, who had contracted the disease, staggered and fell at his side while accompanying him, and died on the eighth day. Though loss of muscular power is not usually so sudden, the patient will be compelled to take his bed within twenty-four hours, and the attending prostration is more marked early than in any other febrile disease. Soon, in the majority of cases, the patient becomes so weak as to be unable to turn in bed, and lies helpless on his back. Paralysis of the sphincters soon attends, with involuntary evacuation of urine and feces. Dysphagia, partial aphonia, and inability to protrude the tongue, are often present. Muscular tremors, subsultus tendinum, picking at the bedclothes, hiccough, strabismus, and opisthotonos, may occur in desperate cases.

The fever may run a typical course, the stadium being reached in many cases as early as the third day. However, this stage may not be attained until two or three days later. The regularity with which typhoid fever advances during the first week is not observed in this form, and a record of the temperature during the first few days would not be much assistance in diagnosis.

Temperature.—Though chilly symptoms may persist for two or three days, the *temperature* rises rapidly, and within the first twenty-four hours may reach 105° or 106° F. In other cases, two or three days may be occupied in the development of the fastigium. The morning and evening variations are most marked at midnight, but these are not regular as in typhoid, there hardly being a regular periodicity, and the crisis occurs without any increase in the length or degree of the diurnal variation. Sometimes, on the day preceding the crisis, the temperature rises three or four degrees higher than before. The temperature usually ranges the highest during the second week.

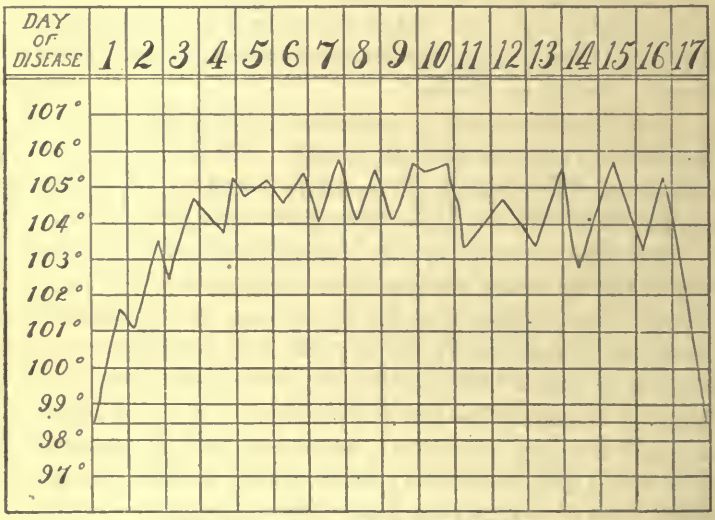
The *headache* of typhus is a notable feature, appearing early, and steadily increasing in severity for the first week. Associated with this is dullness and confusion of intellect, sometimes vomiting. It is said that the headache of typhus is much more constant and severe than that of any other fever.

The *pulse* is rapid and full in the beginning, but it soon becomes feeble, soft, and compressible, and increased in frequency. In unfavorable cases, when a fatal termination is nearing, it becomes

dicrotic, irregular, and intermitting. The *tongue* is swollen at first, and covered with a white coating, but in a few days it becomes brown, and, later, black, dry, and fissured. In severe cases, the tongue is shrunken, and rolled in a ball, at the back of the mouth.

The *countenance* presents a peculiar appearance, the face being darkly flushed, and the expression dull and weary, the cheeks often being of mahogany color. The sleep is restless and disturbed, and, when the patient is awake, his mind does not seem clear, even in the early part of the disease.

Delirium of pronounced character comes on about the eighth day, though it may be present much earlier, and the headache, which has been such an unpleasant feature, now subsides. It is more marked at night, at first, usually passing off in the morning, to return at night; but it soon becomes continuous, varying, in different



TEMPERATURE IN A CASE OF TYPHUS FEVER.

patients, from a low muttering, to the most active and noisy kind. Loomis asserts that acute delirium is most apt to be present when the patient is intelligent and highly cultured, and that of muttering character in aged persons, or those of little culture. Stupor or somnolence follows the period of delirium, the patient lying in a condition of coma vigil, for hours before death. Favorable cases are more apt to be marked by somnolence, prior to the period of crisis.

The *rash* of typhus fever appears from the fifth to the eighth day of the disease, usually on the fifth. It consists at first of dirty pink spots, which appear on the abdomen and gradually extend over the body, showing everywhere except on the face and palms of the hands. These spots vary in size, from mere points to three

or four lines in diameter, are slightly elevated, and disappear on firm pressure. In a day or two the eruption becomes much darker, of purplish, mulberry color, the elevation subsides, and the spots remain under pressure.

Diagnosis.—It is not difficult to distinguish typhus from other diseases which it may sometimes resemble, if it be recollected that it is a contagious disease, confined to its immediate surroundings. While it may resemble typhoid fever in some of its characteristics, it must be remembered that the invasion of typhoid is very gradual usually, while this is sudden in its onset. The abdominal symptoms of typhoid are also characteristic, while tympanites and tenderness on pressure are absent in typhus, and diarrhœa is hardly ever present. The rash, on the fifth day, is entirely different in appearance from that of typhoid, which appears later.

The *duration* of typhus fever is also a distinctive feature, the disease terminating by the fourteenth day, while few cases of typhoid, and no severe ones, terminate within three weeks. Cerebro-spinal fever resembles typhus in some cases, but the rash is different in character and the temperature is lower, hardly ever rising over 102° or 103° F., and the disease runs a much more protracted course, in most cases. Where there is meningeal inflammation in typhus, there are many symptoms in common with this disease, such as the intense headache, delirium, rigidity of the muscles, etc. Only those practicing in seaport towns need take the trouble to exclude typhus from a doubtful diagnosis, as the disease seldom or never penetrates to the interior, in this country.

Prognosis.—This is, without doubt, a dangerous disease, and one which is liable to result fatally, with the best of treatment. Unfortunately, Eclectic methods have never been thoroughly tried, so that no estimate from such a standpoint can be made. Those conversant with the disease and the results of treatment, vary in estimating the deaths from one in five to one in sixteen of the cases affected, in different epidemics. The mortality seems to vary at different times, some epidemics being much more severe than others. The surroundings of the patient certainly exert much influence upon the disease, pure air and other favorable influences encouraging a successful issue. When the patient is debilitated in the start, has been addicted to alcoholism, or is the subject of gouty diathesis, his chances of recovery are diminished. Overcrowding or bad ventilation are opposed to favorable results. Complications are often the cause of death, such as cerebral or pulmonary congestion, and some one of these complications may be a peculiar feature of each epidemic; and as this is marked, so the mortality is liable to be

increased. The prognosis is less grave, when the disease occurs in childhood.

Treatment.—Evidently, from what old school authorities assert, there is little to be expected from treatment; that is, from the action of medicines. Loomis asserts that fresh air is the only thing which will neutralize the poison of this disease. He advises placing the patient in a tent or open pavilion so that the air can circulate freely around him, covering him with blankets, if the air be chilly. He asserts that he has seen patients—apparently overwhelmed by the fever poison, so much as to be in a state of coma with high temperature, and apparently rapidly succumbing to the disease—brought from crowded tenement houses and placed in tents, begin to rally within four or five hours, and go on to speedy recovery. This author places more stress upon such plan of treatment than upon any drugs that may be employed. The same writer deprecates the use of alcohol as a stimulant, averring that, though it may seem to benefit at first, it is liable to finally arrest secretion, prevent the elimination of urea, and disturb nutrition, thus lessening the chances of recovery. He indorses the employment of opiates to induce sleep, asserting that the protracted insomnia is of itself sufficient to cause a fatal termination. To reduce the temperature, he recommends cold baths and quinine as antipyretics during the last week, and urges the importance of proper feeding, stating that the patient must be required, and even compelled, to take nourishment, advising the plan, should he refuse to take it without, of pouring liquids down through a rubber tube passed into the stomach, by way of the nose, when the patient clinches his teeth and refuses to receive it. Milk, malted milk, Horlick's food, etc., will answer the purpose, and some should be given regularly and at frequent intervals, as in the treatment of typhoid fever. During convalescence, care should be taken that the patient be not exposed to sudden changes of temperature so as to take cold, or permitted to overexert himself (as this might result in coagulation of blood in the veins) until after the blood has been restored to a normal condition. Moderate exercise in the open air is commendable. Convalescence is usually rapid.

I am of the opinion that this disease could be much modified by the action of echinacea. I have treated a few cases that I diagnosed as typhus fever at the time, which I am now convinced were cases of cerebro-spinal fever; but from the close resemblance, and the excellent action of echinacea in these cases, I would expect good results from it in typhus; and salicylate of ammonium ought to reduce the temperature and lessen the tendency to blood depravation.

Should the opportunity arise, I hope that some of our Eclectic

physicians will test these remedies in this disease and report results to our medical journals. Doubtless, the antipyretic influence of jaborandi and other vascular sedatives, cold packs, baths, etc., will ameliorate the severity of the symptoms, lower the maximum temperature, and assist in preserving vital structures against destructive action.

III. RELAPSING FEVER.

Synonyms.—Spirillum Fever; Famine Fever; Hungerpest.

Definition.—An acute, contagious febrile disease, characterized by two paroxysms of high fever of from five to seven days each, with an intermission between, of from three to five days' duration.

Etiology.—When relapsing fever occurs in this country, it is the result of importation from Europe. This is of rare occurrence, so much so that the disease has not been deemed worthy of mention by all American authorities on practice. The disease occurred as an epidemic in New York City, in 1872–3, and in Philadelphia, in 1844. In both instances, it was brought from Europe by emigrants. It is highly contagious, and within a few years past bacteriologists have asserted that the producing factor is a parasitic organism—the *spirillum Obermaieri.* It has been called "famine fever," but those who are well fed are as susceptible to the contagion as others. Bad water and food, overcrowding, and vitiated air, predispose to epidemics. The disease is not likely to be carried on clothing, and is seldom communicated except by direct transmission.

The *spirillum* of relapsing fever (spirochæte of Obermaier) is a narrow spiral filament, which measures from three to six times the diameter of a red corpuscle in length, and is readily seen moving about among the blood-disks during the paroxysms—the only periods in which they are visible. Shortly before the crisis and during the intermission they are not found, though small, glistening bodies, supposed to be spores, are then detectable. During the paroxysms, inoculation of a healthy person with the blood of an affected subject will propagate the disease. It is also communicable to monkeys, in the same way.

SPIRILLA OF RELAPSING FEVER IN THE BLOOD.

Pathology.—There are no lesions characteristic of this disease alone, the parenchymatous changes due to febrile action appearing here in proportion to the severity of the disease, as elsewhere. The *liver* and *spleen* are enlarged, the spleen frequently being the

seat of infarctions. The cortical substance of the *kidneys* is congested, and the bulk of the organs thus increased, while granular infiltration of the uriniferous tubules, similar to that noticed in other fevers, may be noticed. In some cases, extravasations of blood are found distributed throughout the organ. Extravations of blood may also be found upon the mucous membranes, especially of the *intestines*, *stomach*, and *bronchial tubes*. The *blood* coagulates imperfectly, as in typhus fever, though coagula in the blood-vessels are rare.

Symptoms.—Incubation.—This period may be short, lasting only a few hours in some cases, though it is usually of six or eight days' duration. During this time there are not often any symptoms to suggest the coming onset.

Invasion.—This is usually abrupt, a pronounced chill announcing the commencement of the attack. This is attended by frontal headache of excruciating character, severe pains in the muscles of the limbs and back, nausea, and vomiting. The temperature rises rapidly, usually reaching its highest point within twenty-four hours after the initiation of the disease. It may rise as high as 104° or 106°, and, in some cases, as high as 109° F.

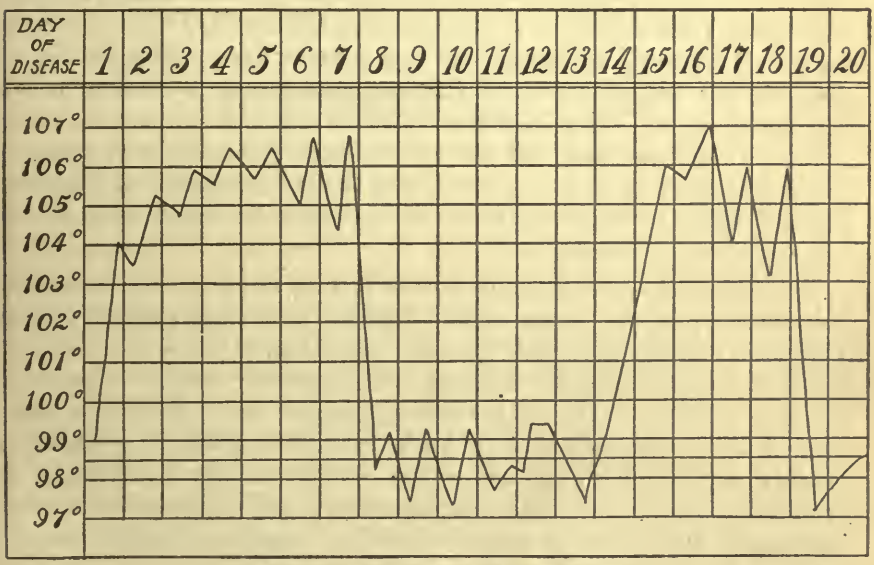
The pulse increases in frequency very rapidly, and this disease is remarkable for the rapidity which the pulse reaches 140, 150, or 160 beats per minute within the first twenty-four hours. It is small and compressible, usually, sometimes dicrotic.

Delirium is not a common symptom, the patient generally retaining control of his mental faculties throughout, though sleeplessness is a common condition, on account of the severity of the muscular pains. The pains may affect the joints particularly, and may become the most unpleasant part of the disease.

As the *liver* and *spleen* become involved—by the second day—weight and uneasiness in the upper portion of the abdomen, especially in the hypochondrium, will be noticed, while enlargement and tenderness of both liver and spleen will be found upon palpation. *Jaundice* develops in many cases, and this may be accompanied by vomiting and diarrhœa.

Marked prostration, irregularities of the pupils, soreness and stiffness of the muscles of the eyes, etc., are other features. By the sixth or seventh day the febrile symptoms have become aggravated to their fullest extent; the pulse is 150 or 160 per minute, the tongue is dry and brown, the muscular pains are excruciating, and emaciation has begun to be marked, while the prostration is extreme, it seeming that a fatal issue must be near at hand. Now, a sudden remission occurs, profuse perspiration breaks out on the surface,

secretion becomes established from the kidneys, the headache and pains subside, sometimes a critical diarrhœa occurs, and the pulse rapidly falls to 80 or 90 beats per minute, while the temperature becomes normal within twelve hours from the first appearance of subsidence. Then, barring a sense of weakness, the patient feels perfectly well. His appetite begins to return, he gets out of bed, and appears to be rapidly convalescing. His pulse will now be found to be slower than normal, and his temperature normal, or near there. But this period of comfort is of short duration. In a few days (three or four, sometimes a week) the attack is repeated, with more severity than before.



TEMPERATURE IN A CASE OF RELAPSING FEVER.

The headache, the arthritic and muscular pains, the high temperature, and the rapid pulse, are again ushered in with great rapidity, sometimes with and sometimes without a chill, and the hepatic and splenic congestion again occurs. This continues from two days to a week, when a second crisis occurs, similar to the former one, and within twenty-four hours from commencement the pulse and temperature have reached normal, and the unpleasant symptoms have again subsided. This time, usually, the convalescence is real, and the patient goes on to complete recovery, though in some cases three or four relapses may occur.

Some of the *complications* of relapsing fever are pneumonia, collapse, ophthalmia, diarrhœa, and dysentery.

Diagnosis.—After the disease has been established, the diag-

nosis is not difficult, its contagious character, the distinct intermission after several days of fever, the severe headache, and the arthritic pains, will afford a correct picture of the disease. Dengue fever, though it resembles this disease in many particulars, is characterized by an eruption, which appears during the second paroxysm, and the remission does not amount to an intermission, as in relapsing fever. In typhus, the headache is almost invariably succeeded by delirium by the beginning of the second week, while delirium is not common in this disease. The intermission occurring by or before the end of the first week will also settle the question of typhus. The slow invasion of typhoid fever will distinguish it from relapsing fever, in which the invasion is accomplished within twenty-four hours after the onset. Abdominal symptoms are also a marked feature of typhoid, while they are not present here. The history of the case will suggest, to the American practitioner, a foreign origin for the disease. Cerebro-spinal fever is marked by severe headache and muscular pain, but the temperature is usually low, as compared with that of relapsing fever, and is very irregular—if high one day it is liable to be low another, and is not to be relied upon to follow any regular course.

Prognosis.—The prognosis in relapsing fever is very favorable, notwithstanding the severe ordeal through which the patient passes. Though a disease attended by much suffering, it does not seem as inimical to life as some diseases of apparently milder character. Loomis estimates that only about three per cent of all the cases treated in the hospitals of New York, during the epidemic referred to in this article, died. Syncope and other complications, such as bronchitis, pneumonia, diarrhœa, dysentery, and uræmia from renal congestion, supply the greatest mortality. Aged and feeble persons may die from collapse during the crisis, though this is not likely to occur.

Treatment.—The Eclectic portion of the profession has had no opportunity to test the value of their methods of treatment in this disease. It is asserted by those who have had extensive experience with it that quinine is of no service in its management. It is also asserted that aconite, arsenic, and veratrum have been tried as antipyretics, without avail. Cold baths have been resorted to with as little profit. It is asserted that opiates have relieved the severe pain, and given better satisfaction than other methods of treatment. Free ventilation should be given the rooms of patients under treatment with this disease, without doubt.

It seems that jaborandi or salicylate of ammonium ought to lessen the maximum temperature of this disease, and, at the same time,

assist in controlling the severe headache and pain. A decoction of rhamnus californica ought also to assist in relieving the intensity of the pain, if not in banishing it altogether.

Complications should be treated as they arise, according to modern Eclectic methods. A liquid diet should be employed throughout the disease, and hygienic methods of management strictly observed.

Of course we will not lose sight of the proper precautions necessary to counteract any tendency to blood depravation which may be manifested by the condition of the tongue. The proper correctives are well known to all modern graduates of our schools, and need not be mentioned here. During convalescence, cactus grandiflorus, cereus bonplandii, or digitalis may be administered for the tendency to heart failure; and this may be necessary, during the first remission.

IV. CEREBRO-SPINAL FEVER.

Synonyms.—Cerebro-spinal Meningitis; Spotted Fever; Petechial Fever; Malignant Purpuric Fever.

Definition.—An acute, infectious disease, characterized by inflammation of the cerebro-spinal meninges; excruciating pain in the head, back, and limbs; irregular fever; and often by convulsions or opisthotonos, and a petechial rash.

Historical Note.—This disease was first identified in Geneva, in the early part of the present century. Soon afterward (1806) it appeared in Massachusetts, and has since visited almost every part of the United States and Canadas.

Etiology.—The exciting cause of this fever is yet a question. It may occur epidemically or sporadically, but is most liable to occur as an epidemic. All ages are subject to it, but young persons and children are most liable to attacks. Bad hygienic surroundings are predisposing causes, overcrowding, bad ventilation, insufficient or unwholesome food, dampness, etc., being supposed adjuncts.

Recent investigations have resulted in the conclusion that the exudate invariably contains a lance-shaped coccus, identical with the diplococcus of pneumonia, and the constant presence of this bacterium suggests it as the exciting cause.

It is not considered contagious, either directly or through fomites, though crowding of communities together, as in garrisons and barracks, seems to predispose to outbreaks.

Pathology.—The pathology of this disease indicates two forms of cerebro-spinal meningitis. The first to be mentioned is the *sporadic form*, where the anatomical lesions are confined to evidences of simple inflammation of the meninges of the brain and spinal

cord, the second or *epidemic* form being characterized by evidences of grave *visceral* and *sanguineous* changes, akin to those of typhus, typhoid, and other putrid fevers. The *brain* is always found more or less involved, the dura mater being tense and shining, and the surface, especially at the convexity and base, studded with punctate points of extravasation. Hyperæmia of the pia mater is also a constant condition, the vessels usually being injected, and the surface roughened, this condition involving both the brain and spinal cord. The sinuses of the dura mater may contain much softly coagulated blood, especially in the epidemic form of the disease, and extensive exudation of sero-fibrinous or sero-purulent fluid is found over both the convexity and base of the brain. In the latter situation, the cranial nerves are often imbedded in this substance. The amount and color of this exudation vary, it sometimes presenting a whitish, soft appearance, and being in small quantity, while again it is abundant, and yellowish or greenish in appearance, suggesting purulency. This condition may involve the posterior surface of the *cord*, a purulent fluid being found under the arachnoid. The *blood* is dark and tarry in appearance in the epidemic form, the fibrin diminished, the white corpuscles increased, the fluid rapidly decomposing when exposed to the air. The *muscles* are dark colored, and the tissues generally have undergone granular degeneration. There is often congestion of the lungs, liver, and spleen, the parts being enlarged and increased in friability. The *skin* is frequently the seat of an eruption, of petechial spots, though there is no regular time for its appearance. After death, purple or purpuric spots appear, especially along the region of the spine; and these are often present during life, in certain epidemics.

Symptoms.—The symptoms of this disease are of wide diversity of character, though there are a few—such as severe headache and pain in the back and extremities—which are invariable. The pain is notably severe in the upper portion of the spine, the head being thrown backward to relieve the tension on the ligamentum nuchæ early in the onset, the suffering here being described as excruciating. The length of the period of incubation is not known.

Invasion.—In some cases the invasion of the disease is abrupt, the patient being seized with a chill and loss of consciousness, while coma, convulsions, and death, may follow within a few hours. But these are the extreme cases in the epidemic form. When the disease is sporadic, the symptoms are more gradual in their advance, and the chill is not so apt to be pronounced, though the patient may complain of chilly symptoms for several days. The reaction is slight, the temperature not rising much above normal. The *headache* and

pain along the *spine* however will be marked, the patient will be restless and sleepless, and there will be loss of flesh and emaciation, as the case progresses. Often the *pupils* will be found of unequal size, while the features present a fixed or staring expression. The vaso-motor supply to the face will be involved early, and irregular control of the blood-vessels will give rise to variability in the appearance of the face, it being brightly flushed or presenting a hectic appearance at times, and within a few minutes afterward showing a ghastly pallor, especially after the patient has become prostrated and debilitated. This irregularity in the circulation of the face is a notable feature, in the protracted form of this disease.

In two or three days, sometimes later, *delirium* comes on. This varies widely in character, sometimes being wild and violent, and at other times mild and muttering. In the slow form this may not appear, however, until several days later. Sometimes it is of a maudlin character, resembling the vagaries of a drunken person, and in women, it may resemble hysteria. I recollect a case treated a few years ago, in which the patient, a married woman of about thirty, resisted my attempts to inspect her tongue during the first visit, cried peevishly when disturbed, and acted so childishly that I supposed her husband, toward whom she manifested the same disposition, had crossed her in some way, and that she was working off a fit of sulks, though he assured me that this was not the case. Within twenty-four hours, however, there were retraction of the head, irregularity of the pupils, opisthotonos, and tonic spasms of the extremities, with coma. Hemiplegia, followed by death, resulted within ten days from the time I was first called. In this case the temperature was normal most of the time, and never above 102° F., the extremities being cold continually.

In other cases the delirium is of the most restless character, and the patient will make violent efforts to leave the bed, requiring an attendant at his side constantly. In one case, that of a little boy, the patient struggled to get out of bed, shrieked, at times, and tore the hair out of his head, before his mother (who took great pride in his curls) could be induced to cut it short. With the majority of infants there is constant restlessness and insomnia, there seeming to be intense and persistent suffering (as there undoubtedly is), and this is evidently aggravated by moving or lifting the patient. *Hyperæsthesia* of the skin and muscles is so marked that the least touch or pressure often elicits complaint, and when a child retains its consciousness, it will cry in anticipation, when preparations are being made to move it.

There are many grades of symptoms in this disease, the spasmodic

action of the muscles coming on early in some cases, and not appearing for several weeks, in others. Some observers have divided the cases into the slow and rapid forms. I once witnessed an epidemic where children were the ones principally affected (the disease following measles), in which a little patient would roll its head, fret, and moan, without intermission, five or six weeks, waste away, and finally die in a state of marasmus, apparently suffering intense pain at the base of the brain, one hand keeping a constant motion backward and forward about the mastoid process for days and nights at a time, the suffering being much increased whenever the child was lifted or moved. During this epidemic, some rapid cases developed, and patients died in convulsions and coma within a few hours after the onset. The disease was ushered in usually like a remittent fever (sometimes with symptoms of cholera infantum), but the means commonly employed to interrupt the paroxysms in that disease produced only temporary effect in these cases, relapses soon following.

The *rapid* form of the disease usually prevails during an epidemic, if at all, while sporadic cases are usually of the *slow* form. On account of the typhoid symptoms which attend, many physicians have been in the habit of confounding it with typhoid fever; but there is no analogy between that disease and this. There are no abdominal symptoms, and there is not the regularity about the temperature, as in typhoid.

In sporadic cases occurring in this country, there is not a very marked change in the appearance of the *tongue*, except during the late period of severe cases, it then becoming pinched, dry, and brown. Usually, there is a remarkable absence of disturbance of the alimentary canal. The tongue is not much altered in appearance, there is no gastric irritability, and the bowels are not disturbed; though there may be slight constipation, but not more than might result from protracted recumbance in a state of health. Sometimes, however, dysenteric symptoms are present, especially if the disease occurs during the heated season. In one instance occurring in my experience, an epidemic was characterized by the appearance of muco-enteritis among children, this afterward becoming complicated with cerebro-spinal symptoms, which soon became prominent.

The *pulse* may be only slightly accelerated, or it may be very rapid. Like the temperature, it is liable to marked variation in a brief period, the pathological changes likely to occur in the neighborhood of the vaso-motor and pneumogastric centers suggesting the probability of such a state of affairs. As to quality, the pulse is small and wiry, in the majority of cases, but becomes dicrotic, late in the course of the disease.

The *temperature* is variable, but there is usually a tendency toward a low range. It is hardly ever above 103° F., and is more apt to range below than above this limit. In many cases, the extremities are cool throughout the course of the disease, and the temperature of internal parts not much above normal. However, in exceptional cases a very high temperature range may be registered early, and usually, shortly prior to a fatal termination, there is marked elevation, even though there has not been much fever before. A record of the temperature in one case is no suggestion as to that of another; the pathological changes occurring so near the heat center seem to disarrange all calculations tallying with experience gained in certain other fevers.

The *eruption* is usually limited to the face, neck, and lips, though it may appear on the trunk and limbs. Vesicles resembling fever-blisters appear on the lips, and may be limited to this region. In other cases, the eruption may resemble that of typhus fever. Ecchymoses may appear on the body, especially about the hips and dorsal region where decubitus has caused pressure, and these are particularly noticeable after death. There is no regular time for the eruption to appear, and no stated length of time for it to remain, it being present throughout the course of the disease in some cases, and only for a single day in others, while in still others there may be all grades between these limits. Epidemics have occurred in which the *ecchymoses* were so prominent that the disease was termed "spotted fever."

The *senses* are markedly affected in this disease. There are photophobia, perversion or loss of taste, and deafness. The patient may stare at one when he is spoken to, or seem to, but make no reply, for the reason that he does not hear what is said; and he may not recognize the presence of any one, when apparently looking squarely at his interlocutor.

The *respiratory tract* is often affected, there being sighing inspiration, in some cases, and in others irritation, amounting to bronchitis or pneumonia. Many other *complications* may arise.

Diagnosis.—This disease is readily diagnosed from typhoid fever by the irregular temperature and absence of abdominal symptoms. It resembles typhus in many of its phases—that is, many cases do—but its non-contagiousness and history will prevent mistake in this direction. In malarious districts it may at first be mistaken for malarial fever, but its persistence, the early development of delirium, opisthotonos, and other nervous phenomena, and refusal to yield to anti-malarial treatment, will soon settle the question. It may be confounded with acute rheumatism occasionally, but the

absence of acid sweats, of swelling of the joints, and the presence of rigidity of the muscles, and cerebral symptoms, will soon exclude this disease.

The greatest difficulty will be in differentiating between sporadic cases of this disease and tuberculous meningitis, where there is absence of tuberculous material in other situations, although retraction of the muscles of the neck, and spasms of the muscles of the extremities, are not nearly so marked in the tuberculous form.

Prognosis.—This is always *grave*, whether the disease occur epidemically or sporadically. It is a disease subject to sudden and repeated relapses, and the mildest cases are liable to finally terminate fatally. Pathological changes occur so near the vital spot,—the medulla oblongata,—that unexpected extension of inflammatory action may ensue at any time, and fatally involve vital function. Children and elderly people are the most unfavorable subjects.

The epidemic form usually lasts about fourteen days, if death does not occur earlier, but an intermittent form may be subject to several apparent relapses, and continue for six or eight weeks.

Serious *sequelæ* are liable to follow recovery, especially among children, such as deafness, blindness, and impaired mental power amounting sometimes to idiocy. Paralysis of the lower extremities may result, with slow recovery, several years being consumed.

Treatment.—The most approved hygienic treatment should be adopted in the management of epidemics of this disease. All disease-producing causes should be removed, such as bad air and improper food and water, and the patient should be placed in a dark, cool, well-ventilated room, away from all noise or cause of disturbance. The food should be liquid in character, such as milk, malted milk or lactated food, and this should be administered regularly, and at frequent intervals, throughout the course of the disease. To quench the thirst, cold water may be given freely. Attention must be paid to the evacuations, and catheterization resorted to if there be retention of urine. Enemata may occasionally be required to evacuate the bowels.

There is little to be expected from ordinary medication in the treatment of this disease. Remedies which ordinarily relieve muscular pain produce no alleviation here, as the pain is the result of pressure on the roots of the sensory nerves, and ordinary analgesics are as good as thrown away when administered. As much may be said of the special-sedative plan of treatment. The point of irritation is so near the vaso-motor center that therapeutic action here is overpowered by the pathological condition, and aconite, veratrum, gelsemium and jaborandi are usually powerless to control

febrile action. Indeed, there is usually little call for this class of agents.

The employment of cold baths and packs, ice-bags to the head, and other depressing local agencies, seems incompatible with good judgment, when we stop to consider that we are dealing with a disease in which there is little reactive tendency, and, almost invariably, a low temperature. The treatment ought to be gently stimulating, and supporting throughout—not depressing in the least.

Opiates should not be administered, as they arrest secretion, debilitate, and lessen chances of recovery. It is better to restrain the patient by force when necessary, using precaution not to annoy or irritate him unreasonably, until a curative agent can have time to act; and, from the nature of the case, this will be but slowly.

The inflammatory action in this disease is not that of simple character, but is probably akin to an erysipelatous condition, where not only sedatives, but remedies which correct an underlying blood-dyscrasia, are demanded. On this theory, I have administered jaborandi with some benefit, but have afterward found it usually unreliable. Later, I began the use of *echinacea*, and now believe that I have found the best remedy extant for this disease. I cannot promise that it will cure every case, for I have lost patients with it who have had the remedy from the very beginning, and so have some of my professional friends. But it is, after all, the only remedy I have ever seen administered in this disease, with the exceptions of jaborandi and rhus tox., which has ever seemed to be of the least benefit. Some cases will recover if left to good nursing, and under these circumstances claims may be made in favor of any plan of treatment; but as some epidemics are light, a low mortality rate would not impress my mind favorably toward ordinary methods of medication.

But if *echinacea* be administered faithfully throughout most cases, and cathartics and opiates be avoided—as well as sedative medication—the mortality will be found to be very small, except in epidemics of the most violent character. Of course this comprehends the best nursing that can be had. Appropriate food, good management, proper bathing—though not too much of this—are required; and other sick-room needs must be attended to. Ten or fifteen drops of a good preparation should be administered every hour to an adult, the dose for children being regulated to correspond. Let this be continued throughout the disease.

Rhus tox., in minute doses, may relieve the restlessness, to a limited extent, and it also relieves thirst and controls nausea. Where frequent convulsions occur in infantile cases, it is the best remedy

we have to combine with echinacea, as a cerebral calmative, and antispasmodic. Where there is marked elevation of temperature with sthenia, *jaborandi* will answer better in controlling the convulsive action and restlessness. The usual doses will be proper here—fifteen or twenty drops of rhus to four ounces of water, dose (for an adult), a teaspoonful every hour. Two fluidrachms of specific jaborandi to four ounces of water, dose, a teaspoonful every hour.

Convalescence is usually slow, and care should be observed during this period to prevent the patient from taking much exercise or being seriously disturbed mentally, for fear of relapse. One of the best safeguards against such an occurrence is the steady use of echinacea throughout, in the usual doses, repeated three or four times daily.

V. SMALL-POX.

Synonyms.—Variola. German, *Pocken; Blätter.*

Definition.—A contagious, eruptive fever, characterized by a peculiar eruption, which is first papular, then vesicular, and then pustular, the disease being further characterized by a secondary fever, which follows the decline of the primary pyrexia upon the development of the eruption, the remission continuing until the beginning of maturation.

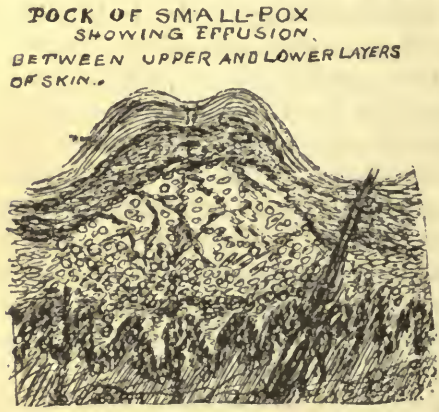
Etiology.—The poison of small-pox is extremely tenacious, no other eruptive fever being capable of retaining its infecting properties in fomites so long. It has made trips across the Atlantic from Europe, and onward across the American Continent, in trunks of clothing, to be afterward liberated, to infect such as were susceptible, who were exposed to its influence. In such cases, the fomites will be found to contain particles of the eruption, in which the virus exists. The contagiousness of such material has been known to remain for years, Goss, for instance, stating that he knows of a case where the disease was communicated to persons while cleaning out a cellar under a house in which patients had been sick with small-pox two years before. It can only be produced by its own contagion, and is only communicable to persons who are not protected from it, such protection consisting in the influence of a previous attack, and, to a considerable extent, in vaccination. A vigorous condition of the system undoubtedly fortifies against it to some extent, it not being so highly contagious as measles. The infectious principle exists in the virus of the pustules, as it may be inoculated from this source, and it may also be conveyed by the breath, as well as by exhalations from the body. In this day, the disease does not seem to be as contagious as it must have been in olden time, as frequent exposures occur without the communication of the contagion. As the period of

maturation of the vesicle is asserted to be the most infectious stage, the time of exposure doubtless explains why so many are exposed who escape the contagion—*i. e.*, the disease does not become highly contagious until the vesicle becomes maturated. Vaccination is also believed to have exerted a generally protective influence upon the whole community, as it was formerly a virulent and rapidly fatal disease, sweeping pestilence and desolation far and wide. It is asserted that during the century preceding vaccination, fifty millions of people died of small-pox in Europe. It is also asserted that the disease is now dying out, and that it only possesses historic interest, as it hardly occurs as an epidemic except in uncivilized lands where the population is unprotected by vaccination. Colored races are especially susceptible to the disease, whether it occur in their native land or where they have been transported. In the West Indies, where it was conveyed from Europe by the Spaniards, in 1507, it exterminated whole races of natives; and in Mexico, where it was carried by the Spanish troops, three and a half millions of people died from its effects. It is asserted that wherever the whites and Indians have lived in the same neighborhood since the introduction of vaccination, the Indians have perished in large numbers, while the whites have suffered comparatively slight effects. One attack usually confers immunity, though this is not invariable. The disease occurs most frequently in cold seasons, a suggestion that lack of ventilation predisposes to its effects. Nursing infants enjoy some immunity, but liability grows intense at the end of the first year and continues up to forty, when it becomes less marked.

The specific cause is probably a microörganism, though this has not been identified as yet, after many attempts have been made to discover it. It is believed that when found it will be discovered in the pustule. It is probable, however, that the breath and emanations from the body contain it as well.

Pathology.—The most characteristic pathological change occurs in the skin, and attends the development of the eruption. This begins with the formation of hardened nodules in the cutis vera, occasioned by swelling and proliferation of groups of cells, each nodule being destined to become a vesicle. This cellular change extends throughout the skin and involves the rete mucosum, and a hard, elevated nodule is soon developed. A process of vacuolation, occasioned by necrobiotic changes in the interior cells, soon sets in in this nodule, some deliquescing to form a set of loculated cavities filled with fluid surrounding a common center of structure which remains firm, holding the center down. These cavities are filled with a serous fluid containing red blood-corpuscles and leucocytes.

As proliferation of cells and accumulation of fluids continue, the tense border around rises, leaving a central, or *umbilicated depression.* Some assert that the center is held down by a sebaceous gland or hair follicle, while others aver that the depression is occasioned by the remains of undissolved fibrous tissue in the nodule. However this may be, the condition imparts a characteristic appearance to the eruption for some time after fluid has appeared in the vesicle, none other of the exanthematous fevers presenting such an appearance during eruption. It is seen that each vesicle is a compound one, consisting of a multilocular aggregation of fluid-cavities around a common center, separated by delicate partitions, and if one of these be pricked and evacuated, the others remain filled, unless too much violence has been employed. The fluid filling these vesicles is at first clear, but it soon becomes opaque, and purulency rapidly ensues, the vesicle being converted into a yellow *pustule,* the structure holding down the center now becoming softened and giving way, allowing the umbilicated depression to rise and present, as the apex of a cone-shaped eminence. An areola of hyperæmic tissue now surrounds the base of each pustule, and when they are closely set, the entire surface of the skin is reddened and congested. A drying up of the pustules is followed by the formation of scales, which dry most rapidly in the center, thus contracting and becoming depressed, here constituting a *second umbilicated stage.*



The *crusts* consist of dried pus-cells and epithelial detritus. After a time these are thrown off by the ordinary exfoliative process. The suppurative action invades the true skin more or less deeply, sometimes perforating it, and invading the subcutaneous structures. Sloughing of the openings follows, leaving cavities which heal by cicatrization, cup-like depressions resulting; and a permanent pitting of the skin is the final effect, when the cutis vera is deeply involved. The mucous membrane of the upper air passages, mouth, fauces, and œsophagus undergo modified changes of this character, and the organs involved are hyperæmic, inflamed, and more or less ulcerated.

The *tissue-changes* common to protracted pyrexia are more or less marked in nearly all the organs. In fatal cases, the blood is dark and lacking in fibrin; there are clots in the right ventricle of

the heart; hemorrhagic extravasations are scattered about beneath the mucous and serous membranes; the heart, lungs, liver, spleen, and other internal organs are softened, and either pale, flabby and swollen, or congested. The mucous membranes are congested, softened, ulcerated, their epithelium partially separated and covered with a tenacious mucus, with here and there evidences of pustulation, in small round spots covered with a false membrane or presenting signs of superficial ulceration. Peyer's patches are sometimes congested, and the pleural cavity may be filled with serous fluid.

Symptoms.—The symptoms of this disease vary so widely in different cases that it will be best to give a general outline first, and particularize afterward.

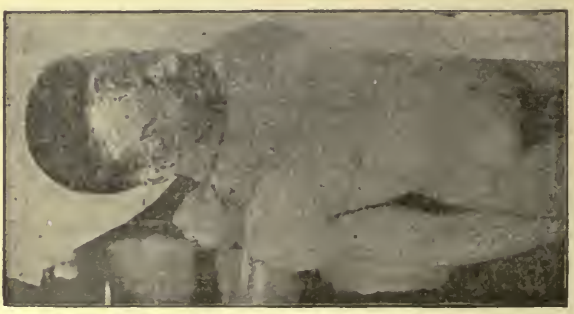
The period of *incubation* varies ordinarily from seven to twelve days, though in exceptional cases it may continue as long as three weeks. During this time it is unusual for the subject to complain of unpleasant symptoms.

The *onset* of small-pox, even in mild cases, is usually abrupt and severe. There is a marked chill, often nausea and vomiting, fever, headache, and *excruciating pain in the loins*, this sometimes amounting to a condition of temporary paraplegia. In children, there may be convulsions at the start, with intervening coma. The febrile action is usually high, the temperature reaching 103° or 104° F. in a few hours, and ranging as high as 105° or 106°, by the time the eruption is out. However, the temperature is often much lower, and it may not reach more than 102.5° elevation during the invasion stage. The tongue is usually coated with a white covering, this often being of a dirty, pasty character; the pulse is accelerated and the skin is moist, perspiration usually being present throughout the stage of invasion. Soreness of the throat will now be complained of, the voice being hoarse and husky, and the patient will complain of pain in the pharynx and difficulty of swallowing. The headache, which is severe in the beginning, gradually increases until the eruption appears, when it subsides along with the fever, backache, and other unpleasant features of the invasion stage. The stage of invasion lasts about three days, and during this time the patient becomes considerably prostrated, often being unable to rise from bed; and he may be extremely restless, and sometimes delirious. During this stage, the menses appear, in the majority of women affected, whether it be at the proper time or not.

The stage of *eruption* begins when minute red points make their appearance along the edge of the hair on the forehead, on the chin, and other parts of the face. This may be preceded by a rosolous rash, which appears during the invasion, upon the inner aspect of the

arms and thighs. The eruption appears on the scalp first and spreads to the face, then appears on the wrists, arms, chest, neck, and other parts of the body, coming out lastly on the lower extremities about twelve hours after its appearance on the face. The papules show a disposition to arrange themselves in groups of threes and fives, scattered more or less thickly over the surface, being most abundant on the face. By the second day, the finger pressed upon them receives the sensation as though a shot were buried in the skin, the nodule at first being firm and resisting. This gradually rises on the surface. Soon the nodule is observed to have become umbilicated, and to contain a watery fluid. The fever, headache, backache, and all other unpleasant symptoms, except the burning and itching of the skin, now subside, and the temperature approaches, though it does not reach the normal standard, and it remains down until the stage of maturation has begun. Vesicles may now be seen in the nares, mouth, and pharynx.

The stage of *suppuration* begins about the eighth day of the eruption, or the tenth or eleventh day of the disease. The fluid in the vesicles becomes turbid from the admixture of pus corpuscles on the sixth day, and by the eighth the stage of suppuration is fully established. A marked ring of tumefaction now surrounds the base

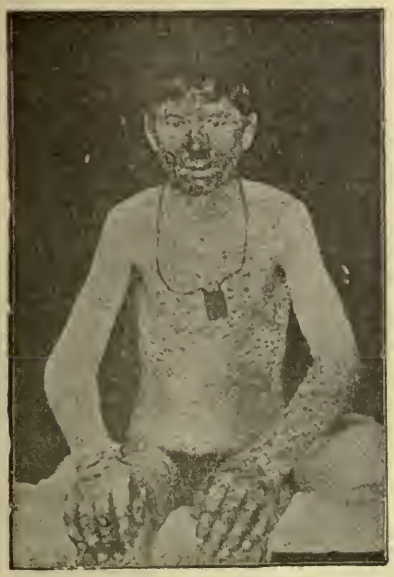


Stage of Suppuration in Confluent Small-pox.

of each pustule, the tissues being reddened, œdematous, and swollen; where the pustules are thickly set, the entire surface swells remarkably; this is especially liable to be the case with the face and extremities, where the eruption is most apt to be confluent. The eyes are obliterated, to all appearance, the cheeks and nose are frightfully deformed, while the entire face is covered by a hideous mask of ripened pustules, and the hands and feet are swollen into balls. A characteristic and sickish odor now emanates from the patient, rendering him obnoxious to the sense of smell, and frightful itching urges him to tear and scratch the affected surface, which oozes quantities of purulent material. The throat is swollen and

painful, and sometimes deglutition is impossible. About the eighth or ninth day of the eruption the pustule is fully formed, and the stage of suppuration is completed. This stage is usually ushered in by a chill, and the temperature rises, sometimes higher than during the stage of invasion, though manifesting a distinctly remittent character, rising in the evening, and declining in the morning. A corresponding increase in the pulse-rate attends, high fever with delirium frequently being present. Sometimes there are typhoid symptoms; the tongue is heavily loaded with a pasty, white coating, or is brown and dry; the patient is restless and delirious, lies in a state of coma, or mutters incoherently; the pulse is feeble and fluttering, or quick and tremulous; there is diarrhœa with involuntary evacuation, and general prostration of all the vital forces. By the eleventh or twelfth day of the illness, desiccation begins, and the fever and inflammation subside.

The stage of *desiccation* is occupied in the drying up and casting off of the pustules in the form of *crusts*. This process begins on the scalp and face, where the eruption first appears, and follows the course of the outbreak. The redness, tenderness, and œdema of the skin now begin to subside, and the purulent material becomes blackened and hardened at the apices of the pustules, forming crusts, which become puckered, contracted, and depressed in the center. Gradually, the skin assumes its normal color and appearance between the pustules, and the scabs loosen and separate, each leaving a reddish-brown stain with a sunken center, which remains reddened for five or six weeks and then disappears, or remains permanent as a whitened scar. With the fall of the crusts, the appetite and ability to sleep return, and the patient begins to regain health and strength.



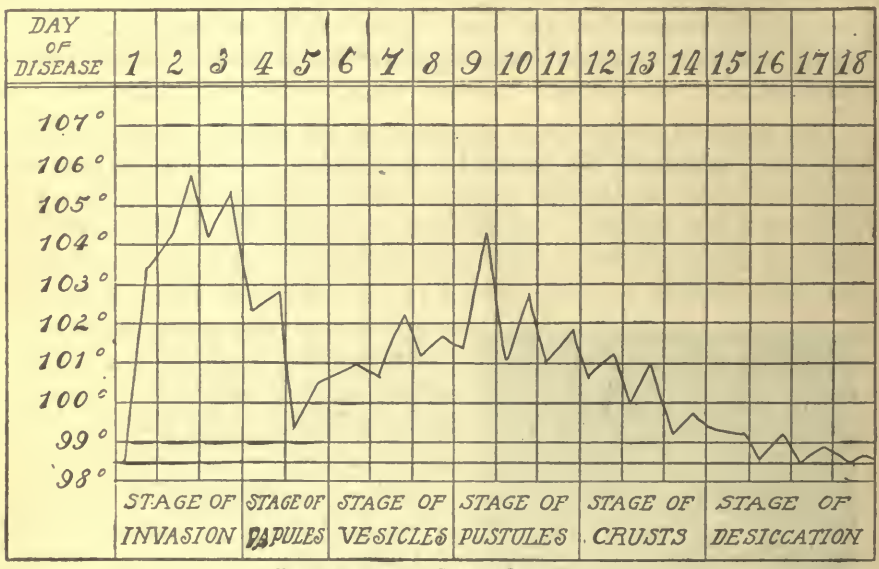
Stage of Desiccation in Variola Confluens.

Authorities on practice usually describe small-pox under three forms, viz., (1) variola vera, (2) variola hemorrhagica, and (3) varioloid.

Variola vera is divided into two divisions, depending upon the amount and extent of the eruption, namely, discrete and confluent.

The *discrete* variety is characterized by a *scattered* eruption, even on the face and hands, where it is most marked. Though the symptoms of the stage of invasion may be severe, the eruption is not attended by severe suffering, and the secondary fever is much modified, as compared with the confluent form, the normal temperature being reached several days earlier, and there being little liability of disfigurement from pitting.

Confluent small-pox.—This is a much more severe form of the disease than the discrete variety. The initiatory stage is shorter than that of the discrete form, and the symptoms are more violent, the temperature rising as high as 107° or 108° F., the pulse being



TEMPERATURE IN A CASE OF SMALL-POX.

correspondingly increased in frequency. The patient may be seized with convulsions, or enter rapidly into delirium or coma, from the very start. Children, especially, are apt to be seized with violent and repeated convulsions. The tongue is loaded with a dirty-white fur, or there is obstinate gastric irritation with persistent vomiting.

The *eruption* appears by the second day, and the red points are closely set, the entire surface being covered early with a reddened tint, interspersed with numerous deep-red points. The throat symptoms are marked and distressing, the patient suffering in the early stage with severe pain in the pharynx, and difficult deglutition, as well as laryngeal irritation, attended by hoarseness and cough. These symptoms are much aggravated during succeeding stages, œdema of the glottis, or extensive sloughing of the tissues of the throat, probably succeeding. The eruption forms large bullæ, with flattened sur-

faces on the face, covering it as a complete mask; and confluent patches may appear on portions of the body, though the confluent eruption is chiefly on the face. The countenance is so swollen as to be unrecognizable, and the suffering is intense, if the patient be conscious. After desiccation, if the patient survive, large, contracting cicatrices mark different portions of the face, the eyes are liable to be destroyed from severe keratitis, and other scarring results. Few recover from malignant attacks of this form. Reaction is not marked in the suppurative stage of this form, the reactive powers being too nearly exhausted; and the temperature may even be subnormal. The pustules may contain blood instead of pus, though this does not comprehend the hemorrhagic form of the disease.

The *hemorrhagic form* of small-pox is characterized not only by the exudation of blood into the pustules, but there are extravasation of blood within the skin and hemorrhages from various mucous surfaces. Here the symptoms of malignancy manifest themselves from the very start, and the patient is liable to die from exhaustion or septic poisoning, before the completion of the stage of eruption.

Varioloid is an imperfect form of the disease, in which the stages are all but faintly marked, and in which the eruption is not fully completed. The disease runs a mild course, and the patient recovers without pitting, and without much discomfort. It occurs in persons who are not very susceptible to the disease, or who have been protected by vaccination. That it is genuine small-pox, so far as the infectious principle is concerned, however, is attested by the facts that an attack affords immunity from visitations of small-pox, and that a susceptible and unprotected person may contract variola in severe form from one affected with it. Many of the cases of small-pox that are met in modern time are nothing more than varioloid, the almost universal resort to vaccination having partially, at least, protected nearly every one from its olden-time ravages.

Complications.—The severe complications which formerly attended this disease are not so common in these days, probably because the treatment has become more rational, as well as the affection less severe. Probably the most numerous of these occur in hospitals, where many patients are congregated. Severe cases, however, are liable to be complicated with destructive inflammation of many different parts and organs.

Pulmonary complications are probably the most serious and frequent. Œdema of the glottis and asphyxia, unless tracheotomy be performed, frequently occurs in severe cases. Severe bronchitis, with extension of the inflammation to the small tubes, is likely to occur in children. Pneumonia not unfrequently occurs, and effusion

into the pleural sac from involvement of the pleura, is then apt to follow. A sequel to severe pulmonary inflammation may be *pericardial inflammation*, resulting in hydrops pericardii or fatty degeneration of the walls of the heart.

Cellular abscess involving the cutaneous tissues may result from the extension of boils through the skin, in scrofulous children. In such cases extensive gangrene may attend the suppurative action.

Sloughing of delicate parts, such as the scrotum and labia, may occur in children, especially those of scrofulous tendency, and deep-seated abscesses may be attended by phagedenic ulceration, upon their discharge.

Inflammation of the conjunctiva, with severe ulceration of the cornea, often with destruction and evacuation of the humors, was once quite a common complication.

Otitis, with destruction of the structures of the internal ear and extensive ulceration of the osseous portion, is among the possibilities of severe cases of this disease.

Suppuration of the joints; destruction of the hair follicles, rendering the patient permanently bald; *bed-sores; meningitis;* and *paralysis*, may be looked for in very bad cases.

Diagnosis.—The history of the case may throw some light upon it, where a diagnosis is called for early in the disease. Is the patient an immigrant, recently from some public route of travel, or has he been exposed to the influence of fomites of such character? Or, is there any account of suspicious eruptive fever in the neighborhood? The fact that the patient has been vaccinated does not militate very much, as its protection may have passed away. The onset of variola is sudden, abrupt. The temperature rises rapidly, the pain in the loins is excruciating, and the tongue is coated white, and not of the strawberry character of the scarlatina tongue. The eruption is also much more "shotty" in feel than measles, and it is more macular in appearance than scarlatina; not so punctate. The catarrhal symptoms are not so marked early in measles, and the eruption does not spare the nose or region of the mouth, as does scarlatina. When the vesicles are developed, there can be no mistake.

Prognosis.—In these times, the prognosis of small-pox is much more favorable than formerly. If treatment be at all modern, few cases take on the severer form, and the pitting and other sequelæ are slight. Unfortunately, most cases are left to the care of the health officer, who is usually of the kind which employs the most unsuccessful therapeutic measures; but still the mortality is usually low after all, owing to the modified form in which the disease generally appears.

When the confluent form is present, the prognosis is much more grave; and hemorrhagic variola is exceedingly fatal, very few recovering. There are so many complications of serious nature attending confluent small-pox, that death from exhaustion is liable to occur during the third week, after the infectious disease has run its course.

Age and sex determine something in regard to mortality; infants and old persons are bad subjects, and likely to die if the disease is severe. Women do not endure the disease as well as men, probably owing to the almost inevitable menstrual disturbance. A severe invasion is not always indicative of a confluent eruption and serious after-effects, as the eruption may be slight, and the remaining portion of the case mild.

Hemorrhages occurring from several of the mucous surfaces—different organs—may be considered indicative of serious results; or early extravasation of blood into the vesicles, or into the skin.

Pregnancy is an unfavorable condition for a patient with small-pox. Œdema of the glottis is a complication that offers poor hopes of recovery, as tracheotomy is imperatively demanded, and this becomes a serious operation under such circumstances.

Treatment.—The *prophylactic* treatment will consist of *vaccination*, immediately after exposure, or as soon as possible. If this should fail to arrest the disease, it promises to at least lessen its severity. The *diet* should be regulated during the stage of incubation, so that all greasy and stimulating food may be avoided, light, farinaceous foods and fruit only, being allowed. Plenty of *pure air* is important in this disease, and it should be seen that rooms occupied by small-pox patients are well supplied with this essential to successful treatment. Loomis asserts that the most satisfactory treatment employed in small-pox epidemics with which he was connected, was administered in barracks, where there were snow-drifts on the floor of the wards occupied by the patients. Pent-up air is quite likely to develop bad cases out of those which might otherwise be light, and must be avoided.

The *medicinal treatment* will consist in following out the simplest indications. The morbid changes which occur in the skin, develop the serious aspects of all bad cases, and if these can be modified or controlled, a mild course is assured. Remedies which lessen the initial hyperæmia will modify the later action and ameliorate symptoms, if they do not shorten the disease.

Eclectics will resort to the *special sedatives* early, even before the true character of the disease is fully developed; and this method is the philosophical plan, as it will control, to a certain extent, the

cutaneous hyperæmia. The whole class of remedies of this group—aconite, veratrum, gelsemium, jaborandi—may be applicable, in treating several cases. But wherever there is not gastric irritation to contraindicate it, *jaborandi* will be the preferable one, on account of the directly sedative influence it possesses on the skin. Two or three drachms of the specific medicine, or some other reliable preparation, should be added to half a glass of water, and a teaspoonful should be administered every hour, as soon as the initial fever begins. This will be an excellent prescription to follow until the fever subsides, as its cooling, soothing influence upon the skin, through the systemic circulation, will be highly appreciated by the patient.

But the frequent occurrence of nausea and vomiting will preclude such treatment in many cases, and something more adapted to gastric irritability will be demanded. Here we may expect the best results from *rhus tox.* and *aconite*, in the usual proportions, five or six drops of aconite and fifteen or twenty of rhus to four ounces of water, dose, a teaspoonful, for an adult, every hour.

The *sodium sulphite* tongue—excessive acidity with sepsis, as indicated by the dirty pasty coating—should not be disregarded in the treatment. The nearer we can bring the condition of the patient to a standard of health in a general way, the less severe will be the later stages of the disease. The brown coating on the tongue suggesting *sulphurous acid* may occasionally be present, and the suggestion should be heeded. As an intercurrent remedy, to fortify the blood against septic changes, and also fortify the tissues against suppuration and sloughing, we certainly cannot do better than administer echinacea. Ten drops of *echinacea* should be given every second hour throughout the course of the disease, as it will be almost certain to modify the tendency to pitting, as well as provide against septic changes liable to attend the extensive accumulation of purulent material upon the cutaneous surface.

In no other form of eruptive fever is the use of *baths*, to cleanse and soften the skin, so important as here. From the very beginning, the surface should be sponged frequently, with warm water and unirritating soap, such, for instance, as Loyd's Asepsin Soap, or some mild and cleansing preparation of the kind. A bath of this character should be administered several times a day by sponging the surface, and after the eruption it should be continued, with the free use of some emollient application, to prevent the skin from hardening and imprisoning the pus, during the stage of suppuration.

Pitting of the face is to be avoided, if possible, and to provide against this the skin over the part should be kept especially moist,

and the air and light should be excluded. For this purpose a *mask* should be worn, this being fashioned from a piece of muslin, of appropriate shape, with openings for the mouth, nose, and eyes. This should be wetted every hour, in a dilution of an ounce each of specific echinacea and glycerine, in six ounces of water. Each time the mask is removed for saturation, the face should be well sponged with tepid water rendered slightly alkaline with mild soap. Or, the following solution may serve better to control the itching: ℞ Resorcin ʒi, glycerine ʒss, water ʒvi, M. Apply with soft sponge, every hour.

Muscular pain, during the initiatory and eruptive stages, may demand special attention. *Jaborandi* is a very applicable remedy, and, as it has been recommended for the initial fever, it will meet this indication as well. However, sometimes we may find it an assistance to resort to *cimicifuga* or *phenacetin*. *Rhamnus cal.* may be found to answer well here, though where there is any tendency to diarrhœa it would be better to depend upon some other remedy. The backache may also be much relieved by the application of hot cloths to the part, though the treatment for muscular pain will probably prove sufficient.

Complications must be met with appropriate treatment. Œdema of the glottis will demand prompt tracheotomy. Pneumonia should be treated by packing the chest with cloths wrung out of tepid water, in addition to proper internal agents, though echinacea will be as appropriate as any remedy in this case. Boils and abscesses should be opened early, and well cleansed with diluted tincture, or specific echinacea. Mouth and throat complications will be pretty well provided for by the general treatment.

There may be times when a *stimulant* may be demanded to prevent fatal collapse, though where echinacea is used properly throughout the course of the disease alcoholic stimulants will be rather inefficient, provided it should fail to sustain the vitality. However, should signs of sinking occur, the judicious exhibition of brandy or whisky is regarded as good treatment in some quarters, and certainly cannot be objected to in desperate cases. However, alcoholic stimulants, usually, should be tabooed, as their stimulating effects are calculated to aggravate inflammatory action and its later result, suppuration.

During the stage of desiccation, daily baths of warm water will assist in softening the crusts and render the skin more soft and pliable. After each bath the skin should be well oiled, the inunction assisting in the process of desiccation, and also acting as a protection to the weakened cutaneous surface, guarding against chilling of the

cutaneous capillaries. The diet should be mild and unstimulating, but nourishing and assimilable.

Supplemental Therapeutcis.—Some think highly of inaugurating the treatment with a thorough alcoholic *vapor bath.* This is doubtless excellent to relieve the lumbar pain, while it may assist internal agents in modifying the entire course of the disease. A thorough *emetic* may be employed with good results in malignant cases marked by drowsiness with tendency to coma, and cold extremities with feeble pulse. Some practitioners believe in small-pox *specifics.* Two agents prominently recommended by their respective admirers are *cimicifuga* and *sarracenia purpurea*—pitcher plant. In the use of either of these a decoction is preferred, the dose being a tablespoonful, repeated every three or four hours.

VI. VACCINATION.

Definition.—The introduction of cow-pox into the human system, as a protection against small-pox.

History.—In 1776, Dr. Edward Jenner observed that in some of the northern counties of England, employés of the dairies there who suffered from a certain form of ulcer upon their hands apparently contracted from cows while milking, possessed immunity from small-pox. Like many other medical discoveries, however, this fact was known to the people a long time before Jenner noticed it, and his attention was probably first called to it through this medium. History has it that a Holstein schoolmaster vaccinated three pupils in 1771, and in 1774 an English farmer vaccinated his wife, because of his belief in the power of bovine virus to prevent small-pox, as seen in his dairy-maids.

Jenner made his first vaccination on a man in 1796, and published his belief in the doctrine first in 1798. Waterhouse, of Boston, introduced the practice into this country in the following year, and in 1800, it was introduced into France. For the first six years after the announcement of his discovery, Jenner was subjected to the most outrageous villification and abuse imaginable by his countrymen, all over Great Britain. He was attacked by the leading physicians and surgeons, reviled and denounced from the pulpit by the clergy, and scoffed at, as the "crack-brained doctor," by the common people. Placards, containing caricatures of Jenner, were posted throughout the principal streets of London and other large cities and towns of Great Britain, and he was treated to many other indignities. Within six years, however, there was a revolution of sentiment, Jenner, by this time, having compelled the profession, by his success, to adopt

his views; and soon afterward, vaccination became generally practiced for the prevention of small-pox.

But the opposition did not altogether cease here. In spite of the fact that almost the whole world was convinced, when small-pox was ravaging Europe and there was so favorable an opportunity to observe the contrast between those protected and those unprotected, a small minority maintained their opposition; and there exists to-day, in England, and to a limited extent in America, a class of people calling themselves *anti-vaccinationists.* They assert that they do not object to the vaccination of others, but they cry out against compulsory laws demanding it upon themselves. They claim the privilege of being let alone, and being allowed to face small-pox without the protection of kine-pox. They dwell upon tales of horrible diseases transmitted by vaccination, such as syphilis, scrofula, skin diseases, etc., and of erysipelas and other serious conditions being transmitted or developed, through the operation. In twenty-six years, I have never seen anything worse than a few mild cases of erysipelas, though there doubtless are exceptional instances where vaccination may result very injuriously. Accidents may sometimes occur in the simplest affairs of life. I once knew a man to die through having a corn cauterized with sulphuric acid; but this need not forever taboo the practice of attempting to destroy corns. The kind of freedom desired by the anti-vaccinatonists would be like that which permitted a man to burn down his own house whether it joined that of a neighbor or not; welfare of the neighbor's house, in his opinion, seemingly, ought to hold no comparison to his own personal freedom. The proper kind of liberty is that which confers the greatest good upon the greatest number. However, no doubt the anti-vaccinationists have been beneficial to mankind. Their outcry has been conducive to greater caution in the preparation and introduction of material for use in vaccination, in order that bad results may be avoided. Non-humanized virus is now largely used, it being obtained by inoculating healthy calves, the management of vaccine farms being followed as a special business. The material is usually furnished to physicians through the drug trade.

However, non-humanized virus soon loses its specific contagium, and, if the material is not of recent origin, it is very liable to fail to produce the desired effect. Even when fresh, it is estimated that it will prove successful in only about 70 per cent of the cases treated. According to my own observation, 50 per cent would be a better estimate. Where several children are vaccinated at the same time in a certain family, and the operation proves successful in one, it cannot be improper to vaccinate the others of the same family from this

vesicle, provided it has developed a normal course; for humanized virus is much more reliable than animal. If symptoms of erysipelas or severe inflammation should develop during its course, there would be good reason for avoiding this virus, and it would not be likely to contain the element of cow-pox infection. It would be a better plan, however, not to vaccinate those of another family, as, if there should be any objectionable taint in the first, it might be conveyed to others, in this manner.

Vaccine virus is now usually supplied to the market in the shape of "points," these being thin slips of pointed bone, the tips of which have been dipped into the contents of a bovine cow-pox vesicle, and dried, for the market.

Vaccination consists in moistening the tip or point of one of these in pure water, and with it, scratching the cuticle away from over an area of about a fourth of an inch in diameter on the arm, above the insertion of the deltoid muscle. The scratching should be done so

STROKES AND CROSS STROKES. BONE POINT FOR VACCINATION.

that one series of scratches will cross that of another, and it should be continued until slight capillary hemorrhage appears upon the abraded surface. When this occurs, the point should be again moistened, and both sides of the portion covered with virus should be carefully and sedulously rubbed upon the abraded surface, until the material has been thoroughly incorporated with the oozing fluid, and forced into the ruptured capillaries. The operation of vaccination from the arm of one subject to that of another, is very simple. A needle may be thrust into the ripened vesicle, and afterward pushed into the skin of the one to be vaccinated. To render the infection more certain, the operation may be repeated, the point being introduced a second time into the first puncture made in the arm of the person to receive vaccination.

If the vaccination passes through the following stages, it may be considered as having exerted a protective influence upon the subject: Upon the third day after the vaccination, there will be developed upon the site of the operation a small red point or papular elevation, which becomes a bluish-white vesicle, and upon the fifth day there will be developed around this a yellow margin. This vesicle increases slowly in size up to the eighth day, when it is seen to be umbilicated. A reddish areola now appears, developing around it, this

showing faintly on the seventh day, and being very distinct by the ninth. This areola continues to increase in size, spreading around the vesicle for three or four days more, until, by the eleventh or twelfth day it may be one or two inches in width from the vesicle, in all directions, and the redness be marked. The arm will now be swollen and elevated about the vesicle, the neighboring axillary glands will be hardened and enlarged, and the arm and axillary region somewhat tender and painful. The pustule ruptures on the twelfth or thirteenth day, and by the fifteenth the crust is found to have assumed a brown color, which deepens until the seventeenth or eighteenth. This falls off spontaneously on the twentieth or twenty-fifth day, leaving a purplish-red scar, which gradually turns white. Meantime, after the rupture of the vesicle, the reddened areola gradually fades away, and the swelling and tenderness subside, until by the time of the fall of the crust the soreness and inflammation have completely disappeared.

If, instead of the vesicle on the eighth day a pustule be formed, a disturbance of the regular development of a vaccine vesicle is announced, and the vaccination cannot be considered as protective. The intervention of erysipelas is very liable to destroy the specific character of the vesicle, and interfere with the protective effect of the operation.

Vaccination should be resorted to during the first year of life, and again every seventh year until puberty; it should then be repeated again, as the protection gradually dies out. After this, and before as well, vaccination should be repeated upon the advent of every case of small-pox into the neighborhood.

VII. CHICKEN-POX.

Synonym.—Varicella.

Definition.—A mild, acute, infectious, eruptive disease, chiefly affecting children, characterized by a vesicular rash involving the superficial layers of the epidermis, attended by slight febrile disturbance, no important sequelæ, and favorable prognosis.

Etiology.—This is a disease of infancy and childhood, in a large majority of cases, though it may occur during adolescence, and even in adult life, in rare cases. However, such a large majority of cases occur during and before the first two or three years of age, that the physician seldom sees it in later life, especially after the sixth year. It is said that infants under six months of age enjoy a certain amount of immunity. It does not occur in marked epidemics, as some of the other eruptive fevers, but is liable to appear in large cities at all seasons of the year and at all times, its appearance seem-

ing to be sporadic in character, in some cases. The disease is contagious, the respired air being the medium of contagion, probably, though it is asserted that it may be conveyed by a third person. Though efforts have been made to isolate the microörganism of this contagion, they have so far been futile. For a long time there was much confusion as to the identity of this disease, many believing it to be a modified form of small-pox, identical with the irregular cases of varioloid which occur after partial protection by vaccination. But the fact that such cases may originate small-pox in the unprotected, while varicella never produces such a result, establishes the identity of this disease as a distinct affection.

Pathology.—The only distinguishable morbid condition arising from this disease is that occasioned by the cutaneous eruption. This consists of numerous minute red spots, varying from twenty-five to two hundred, which soon become small vesicles containing a clear, watery, alkaline fluid. These rest on a hyperæmic base, though in many cases the areola is absent. There seems to be a division of opinion, as to the internal structure of these vesicles, some maintaining that they consist of a single cavity, while others assert that they are divided into compartments by delicate partitions. From other testimony, it seems that some consist of single compartments, and others may be divided. As the superficial layers of the skin only are involved, the structure is so delicate that the vesicles do not bear much investigation without rupturing. They arise from an exudation underneath the superficial layers of the epidermis, lifting them from the rete malpighii, this layer not being involved. The eruption may, and often does, involve the mucous membrane of the mouth and throat; and even the alimentary canal may suffer. Sometimes, however, the deeper layers are involved, and even the true skin may become ulcerated, and pitting result. However, this is more likely to be the effect of scratching.

Symptoms.—The period of *incubation* usually occupies from ten to fifteen days. The *invasion* stage of varicella is more generally absent than observable. Generally the first symptom noticeable will be the rash, and this will appear while the child is playing about, and making no complaint. The physician is then summoned, because the mother becomes alarmed, and desires a diagnosis made of the condition. Again, the rash may be attended by a slight feverishness, which occasions peevishness and irritability. The temperature may be found at 101° or 102° F., though rarely higher, and not often as high as 102°. Still, there may be some complication in other cases, which will occasion considerably more elevation of temperature, such, for instance, as malarial fever. I have seen this

disease complicated in this way and attended by a regular remittent fever, with morning remissions and afternoon exacerbations, the temperature reaching 103° and more. In such cases the eruption may be the source of considerable discomfort from the itching occasioned, when the fever is at its height. Sometimes the eruption in the mouth becomes a marked source of irritation, much smarting and burning being occasioned when the patient partakes of food.

The *eruption* appears at first as small, red, slightly-elevated spots, resembling the rose-rash of typhoid fever in appearance, which come out first on the upper part of the back and chest, and spread rapidly over the body, face, scalp, and extremities. The face, especially the forehead and temples, furnishes the most characteristic and abundant eruption. There is great variability as to the abundance of the eruption, some cases furnishing only a few scattered vesicles, while others cover the entire cutaneous surface thickly. A few hours after the maculæ or hyperæmic spots appear, a small vesicle can be observed in the center of each macula, and this quickly enlarges to its full size. When developed fully, the vesicles are ovoid or round in form, and vary in size from that of a pin-head to that of a small pea. They are thinly covered, being quite superficial, incased only by the outer layers of the epidermis, and the covering is on the stretch, while there may be a slight zone of redness about the base. These are so shiny and glistening in many cases as to resemble drops of water on the skin. They are sometimes congregated into small groups, resembling zoster. The fluid in these vesicles is clear as water at first, and invariably of an alkaline reaction. As the vesicles mature, they may become cloudy and yellowish from the presence of a few pus-cells, but they never become purulent. Fresh crops of maculæ succeed each other by a few hours two or three times, so that the vesicles may be observed in all stages of progress over a limited area, though those which appear first are the most perfectly formed, many of the later masculæ never passing beyond the vesicular stage, but fading away soon after their appearance. Others form small and imperfect vesicles, but these do not arrive at maturity. By the second or third day the eruption begins to decline, the vesicles becoming wrinkled and flaccid, from partial absorption of their contents. Others grow tense and burst, and still others are ruptured by the patient, while scratching. As they dry up, they form thin, brownish crusts. In a few days, the crusts fall off or are scratched off, leaving reddish patches of skin at their sites, which gradually assume the normal tint. In some cases ulceration of the skin occurs, and permanent, pitted scars remain.

If the mouth and throat are examined during the stage of erup-

tion vesicles may be found here, they being most numerous on the palate, hard and soft. These soon rupture, leaving small ulcers, which sometimes become quite irritable. Sometimes the cervical glands are slightly enlarged and tender. The prepuce and vagina may become the seat of vesicles, and when this is the case, painful urination, and smarting following the act, are complained of. A severe diarrhœa occurring during this stage would suggest the presence of the eruption upon the intestinal mucous membrane.

English authors describe a form of varicella which they term *varicella gangrenosa*, and which is characterized by the appearance of gangrene in the vesicles, these spreading and manifesting a tendency to sloughing, instead of drying up, as in ordinary chicken-pox. The gangrenous vesicles are the seat of deep ulcers, which penetrate the skin and attack subcutaneous structures. It is said to be very fatal, pyæmia and exhaustion resulting, in many cases. It seems that the disease is not confined to puny, illy-nourished children, but may attack the robust and well-conditioned. Epidemic influences and unsanitary surroundings may be responsible for it. It has never been reported in the United States, to my knowledge.

Diagnosis.—The diagnosis of varicella would not be such an important matter, were it not for the fact that varioloid and this disease are frequently confounded; and such a mistake might place the practitioner in an awkward dilemma, provided he diagnosed a case of varioloid as varicella, and caused the exposure of a neighborhood to liability of small-pox. And it is a matter of history that for quite a long time varicella was not recognized as a separate disease at all, but was considered as a variation of varioloid. Care should be exercised, then, in doubtful cases, that the physician does not jeopardize his reputation by a blunder in this direction. The imperfectly formed vesicles of varioloid may resemble those of chicken-pox, and confusion arise, unless some other important particulars of development be taken into consideration. It will be remembered that the eruption of varicella appears at first upon the upper part of the body, and spreads from there to the face, while variola, like small-pox, appears first on the forehead and face. The invasion stage of varioloid is also more marked than that of chicken-pox, there often being vomiting, backache, headache, and considerable elevation of temperature (two or three days before the appearance of the eruption), while in varicella, if any period of invasion be noticed at all, it will not continue more than a day before the appearance of the eruption. The age of the person attacked also will enable one to arrive at a pretty positive conclusion, for though varioloid might attack very small children, varicella is a disease that would hardly be expected at all,

to attack adults. An eruption, then, of the character of that of chicken-pox found upon an adult would suggest varioloid, though it would not be positive proof. The maculæ of varicella are soft, and are seen to be merely hyperæmic spots in the skin when the surface is put upon the stretch, while the papules which first appear in varioloid are more deeply imbedded, and impart a shotty feel to the finger when pressure is made upon them. Also, they develop slowly, the papular stage continuing three or four days. It has been asserted that certain syphilitic eruptions resemble varicella; but the multiform character of syphilitic eruptions, their chronic course, and the absence of pronounced fever, ought to distinguish this disease from varicella, to the most superficial observer.

Prognosis.—Varicella is the most benign of all the exanthematous fevers. It is so mild that little need of medication arises in the majority of cases, though unpleasant symptoms may appear, calling for treatment. I have never seen a case which occasioned me the least anxiety, and would consider that one must have been very badly treated indeed, if it did not progress favorably from the beginning. A few permanent cicatrices may remain upon the face, as the result of violence during the stage of eruption, but these will not be so numerous as to constitute disfigurement, and they will occur in but few cases.

Treatment.—The treatment of varicella will not be a matter that will be of much moment to the physician, many times, as his services will usually be required more for diagnostic purposes than for treatment. If a febrile condition is found to be present, with restlessness and irritability, a combination of *aconite* and *rhus tox.*, in water, in appropriately minute quantity to act as a gentle sedative without disturbing the circulation, will be found serviceable. Where the vesicles have appeared in the mouth, and cause unpleasant smarting upon the taking of food or drinks, minute doses of *phytolacca* and aconite will be useful. For a child two or three years of age, two or three drops of Lloyd's aconite and ten or fifteen drops of phytolacca added to four ounces of water, will furnish enough medicine, when a teaspoonful is administered every two hours. Where there is marked periodicity in the fever, one or two grains of *arseniate of quinia*, 3x trituration, may be given every four hours, until the periodicity ceases, which will be in two or three days. Itching of the skin may be quieted with alkaline baths, or the application of the following mixture: ℞ Resorcin one drachm, alcohol one ounce, glycerine half an ounce, and water ten ounces. Mix, and apply with a soft sponge, or linen cloth. Large vesicles upon the face should be emptied early, the openings being well sponged, to anticipate pitting,

and prevent itching, and scratching which might result in scarring. During the course of the disease, an even temperature should be maintained in the room occupied, and the patient protected from draughts of cold air. Physic should be tabooed, as well as the use of stimulating food.

VIII. SCARLET FEVER.

Synonyms.—Scarlatina; Scarlet Rash.

Definition.—An acute, contagious disease, characterized by inflammation of the skin and mucous membranes, accompanied by an eruption of bright-red color (from which the disease takes its name), a high temperature, a tendency to destructive inflammation of the throat, and an unusual predisposition to nephritis and desquamation of the cuticle.

Etiology.—The cause of scarlet fever is a specific infection—presumably a microörganism, though it has never been isolated. It may be communicated directly, from one affected, or through a third person. The epidermis seems to carry and preserve the poison for months and even years, and the disease is most commonly disseminated by desquamated particles, which find lodgment in clothing, carpets, upholstery, etc. Mail-packages, accidentally containing it, may convey the disease for long distances, and travelers may carry it in their baggage, from continent to continent. Another medium of transmission is hair, in which particles may become lodged, and transmitted from place to place. Loomis asserts that an instance occurred under his observation in which the disease was carried by a dog, from the children of one family to those of another, the animal, having been around the infected children for several days, afterward making a single visit to a neighbor's house. The breath of affected persons undoubtedly contains the infective principle, this probably being usually the medium of direct transmission.

The danger to infection of fomites is proportionate to the length of time they are exposed to the contagium. A member of a family where the disease was prevailing, or the nurse, would be more liable to convey it in clothing than the physician, who would only make brief calls. It is rarely the case that the disease is transported by a medical attendant.

Another medium of conveyance is food, especially milk, the dairyman being capable of communicating the disease to many families, should it occur in his own household. Furthermore, it is believed, by some, that cattle may be affected from it. It is inoculable.

A single attack of scarlatina is usually protective against subsequent exposure. The accounts we frequently hear, of several attacks of scarlatina in a single individual, should be received with much

allowance, as it is common among physicians to render a grossly incorrect diagnosis between this disease and rubella. Many physicians diagnose rubella, habitually, as scarlatina, there being considerable resemblance in the general picture; and we often hear of scarlet fever in the neighborhood when there has been none about, but simply slight cases of rubella. This accounts for the assertion that a child has had the disease and is protected, when it is really as susceptible as ever to scarlatina, and falls a victim, on exposure.

It is essentially a disease of childhood, though adults may be affected. Adults, however, usually escape with a mild attack.

Pathology.—The general *tissue-changes* of fever are well marked in this disease, and hardly require special mention. There are the granular degeneration, the loss of fibrin in the blood, the congestion of the brain, spleen, liver, and other internal organs, and other changes due to a protracted pyrexia, which we find, more or less, in all febrile diseases. The *skin* and *throat* bear the principal brunt of the disease, and the results are manifested in the anatomical changes which occur. Often the *kidneys* become involved, and the alterations which attend and follow acute nephritis are manifested in these organs.

Sequelæ result in destructive inflammation of the middle ear, inflammation and suppuration of the glands and cellular tissue of the neck, keratitis, inflammation of the serous membranes, etc.

The *eruption* may fairly be regarded as the distinguishing lesion. The skin becomes excessively hyperæmic, congested, and œdematous, the fingers being swollen and stiff, this being attended by serous exudation into the rete Malpighii. Rapid cell proliferation in the underlying layers of the epidermis results, and when the hyperæmia subsides the epidermis is cast off, the exfoliation being due to the excessive production of newly-formed epidermis beneath. During the period of hyperæmia, extravasations may occur in the skin.

The *mucous membrane* of the *mouth* and *throat* becomes congested, extravasated, and œdematous. An abundant secretion of catarrhal material is usually poured out, forming a tenacious coating upon the surface of the tonsils and fauces, though the parts may be dry and glazed, instead. The color is bright red at first, but in several cases the parts become strangulated and sloughy, with dark and livid color. Follicular sloughing, or a more general breaking down, may invade the tonsils and adjacent parts. The subcutaneous tissues may be involved in suppurative action, giving rise to retro-pharyngeal abscess, and the connective tissues and cervical lymphatics may be invaded, extensive sloughing abscesses resulting, with destruction of arterial twigs, followed by dangerous hemorrhage. The parotid

and sublingual glands may be affected, severe inflammatroy action attending.

The *kidney* presents the characters of acute Bright's disease. The entire organ is congested, extravasations occurring here and there. The glomeruli are altered, and the convoluted tubes are sometimes found to be the seat of croupous inflammation, this involving the entire length of the tubuli uriniferi. All the characteristics of severe acute nephritis may be observable.

Symptoms.—The period of *incubation* varies from a few hours to a week, the time usually being six days. It is difficult to describe the symptoms of this disease understandingly in few words, as there is a great diversity of conditions, depending upon the severity of the disease, the character of the epidemic, the parts most violently affected, and other states which modify or aggravate the character of the attack, in some particular or manner. I will here describe the symptoms of an average case, and afterward endeavor to discriminate between some of the most marked classes of cases. The disease may be divided for description into three stages, naturally marked by their peculiar symptoms, viz., a stage of *invasion*, a stage of *eruption*, and a stage of *desquamation*.

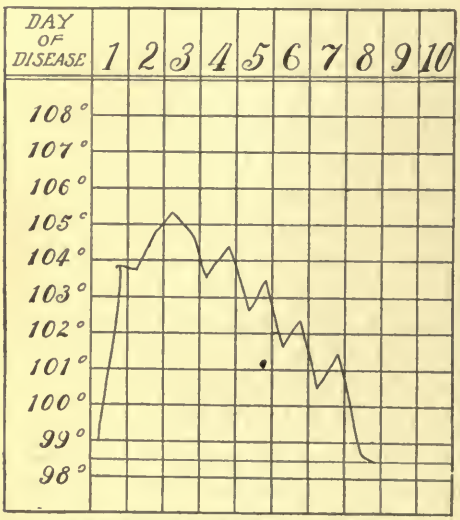
The *invasion* stage begins abruptly. A chill is followed by high fever, though the temperature rises progressively through this stage until the rash appears. The *chill* is usually marked, sometimes amounting to a rigor, and in small children it may be attended by convulsions or coma. *Rapidity of pulse* is characteristic of this disease as an eruptive fever, 130 to 140 beats per minute not being an unfrequent rate. *Vomiting* is almost a constant symptom, the ejection being forcible and projectile in character, and the gastric irritation difficult to control. *Burning* and *prickling* sensations in the skin and throat, with stiffness of the muscles of the neck, are early symptoms, and the skin imparts the sensation of pungent heat to the hand of the observer. *Headache* is marked, being aggravated by the vomiting, and restlessness and delirium may occur, even during the pyrexia of this stage. In one or two days, usually in about thirty-six hours, the rash appears, and the stage of invasion comes to an end.

The *eruption* stage usually begins with the temperature at 103° or 104°, but this soon rises to 105° F., or more. The vomiting, pungent heat of the skin, throat mischief, and nervous symptoms, soon become aggravated; the patient vomits more frequently, is very restless, and often the delirium increases. The rash first appears on the neck, breast, and back, rapidly spreading over the entire surface—except an area about the mouth, where it seldom appears—thus

constituting a rather distinctive feature of the disease, the lips being pallid, the contrast with reddened cheeks thus being quite striking. The eruption first appears as scarlet points (not elevated above the surface) which gradually spread until they coalesce, the borders being of lighter color than the center. They then coalesce, forming an even pinkish or scarlet ground, dotted with minute bright red points. The rash may or may not be confluent, it often appearing in isolated patches, instead of being evenly spread over the surface, it then being found on the chest, abdomen, neck, cheeks, and inner aspect of the arms and thighs. It disappears on pressure, and after the fourth day a letter may be traced upon the surface, to remain until all its outline is completed, before fading out. This is a characteristic peculiar to scarlatina, rashes similar in appearance hardly leaving so permanent a line. The color of the rash may vary, it being scarlet in some cases, in others pinkish, and in still others of a dusky or purplish hue. The last characteristic marks grave cases, and the danger is in proportion to the depth of the purplish tint of the eruption. *Miliaria* often appear about the neck and chest, and the papillæ become prominent in some cases (cutis anserina), while petechiæ mark the points of minute extravasations. By the third or fourth day the eruption has reached its full development, and it begins to fade by the fifth. The skin is now found to be œdematous, the hands and fingers manifesting the effusion most markedly, the fingers being swollen and clumsy from the subcutaneous œdema. By the tenth day the eruption has usually disappeared.

During the *eruptive* stage the general symptoms of the invasion stage are all aggravated. Intense thirst is present. The vomiting often disappears after the first day, though it may continue in an aggravated form, especially if the disease has thus far been clumsily treated. The *tongue*, which in the start was covered with a white coating, except at the tip and edges, which were reddened, now cleans and presents a deep red appearance, with elevated papillæ resembling a ripened strawberry in its aspect. It is moist in moderate cases, but in more severe ones it, as well as the mouth and throat, becomes dry and parched, the mucous membrane presenting a glazed appearance. Later, the tongue may become brown and cracked. The throat now becomes decidedly hyperæmic, the soft palate, uvula, pillars of the fauces, and often the posterior surface of the pharynx, presenting a bright red appearance, the parts being swollen and œdematous. The œdema may be so marked that the uvula and tonsils meet, closing the fauces. When moist, there is a tenacious secretion—exudate from the follicles of the tonsils—adhering about the openings of the follicles, or spread over the inflamed

mucous membrane. In some cases the secretion is absent, the mucous membrane being dry, and the exudation retained, forming abscesses in the follicles. Sometimes retro-pharyngeal abscesses may result from the inflammatory action. Ulceration follows the eruptive stage in severe cases usually, though in very bad ones it may occur earlier, follicular ulceration being the least extensive form, though larger areas may be invaded. The patient complains of severe pain upon swallowing, the voice is nasal in character, the neck is stiffened and swollen, the cervical glands are knotted, hard, and tender, and extensive inflammation of the connective tissue may portend deep and dangerous abscesses in the cervical region.



TEMPERATURE CURVE IN SCARLET FEVER.

The *temperature* of the eruption stage usually rises until the rash is fully developed, when it gradually falls, a crisis generally occurring when it begins to fade, the temperature falling to normal. A slight rise in temperature after this is not unexpected, the cutaneous changes occasioning more or less symptomatic fever, an elevation of a degree and a half or two degrees continuing until the eruption has faded and desquamation has well begun.

The *pulse* continues rapid, as long as pyrexial action exists, *rapidity* being a characteristic, the rate often reaching as high as 160 per minute during the fastigium.

Secretion from the skin and kidneys is arrested, the urine being scanty and highly colored, and usually containing excess of bile pigment, and a sediment of lithates, or free uric acid.

The *desquamative stage* begins a few days after the rash has disappeared, though there is no regularity about the beginning of this period. Sometimes it begins before the rash has entirely faded, and again it may be weeks before desquamation will be noticed. The more intense the eruption the earlier desquamation usually begins, for the pronounced changes in the epidermis tend to a prompt casting off of the superficial layer. In milder cases, the casting-off process is liable to be postponed for a long time—even four or five weeks. Prior to the peeling process, the epithelium presents a dry, wrinkled appearance, after which it begins to break away in fine,

bran-like scales. Over the neck, breast, and other parts where the skin is delicate, it separates in this way; but where it is thick, as in the palms, and on the soles, the epidermis may come away in large patches, even the entire surface of the palm being cast off in a single patch, in some instances. The period of desquamation commonly lasts ten days or two weeks, but it may continue for a much longer time in some instances, relapses of desquamation seeming to sometimes occur, the scaliness lingering about the fingers and toes. It is asserted, by good authority, that the infection lingers until the last scale of epidermis has been cast off. During this period, the pulse is abnormally slow, falling to 65 per minute in many instances, and the temperature is depressed to subnormal. The skin now lacks its normal covering and protection, and the patient is easily affected by a cool temperature, or sudden changes of air.

The great variation in the symptoms of this disease, has led to its division by authors, for description, into three varieties, viz., *scarlatina simplex*, *scarlatina anginosa*, and *scarlatina maligna*. These forms are apt to occur epidemically, the simple form sometimes marking every case, while the more severe anginosa form may prevail in another. Occasionally, the malignant form appears as a scourge, attended by frightful fatality.

In *scarlatina simplex*, the symptoms are all of a mild character. The chill is slight and the reaction mild, the temperature not reaching more than 102° or 103° F., the eruption coming out in patches (with little œdema of the skin) of bright scarlet or pink color. The soreness of the throat is not severe and passes off early, the fever declining in a day or two, convalescence thus being soon established. Desquamation is slight, when compared with that of the other forms. Though this variety is treacherous, the prognosis is usually favorable. Still, each case demands careful management.

Scarlatina anginosa appears in more severe form. The chill is marked, the febrile reaction is high, and there is vomiting and headache upon the appearance of the eruption, which becomes confluent. The throat symptoms are quite severe, though not extremely destructive, and the temperature reaches 105° to 106°, the patient frequently being delirious and restless, or suffering with thirst, headache, and other marked discomfort. Late in the course of this form, ulceration of the tissues of the throat may occur, but it does not seem of that active kind which attacks the throat in malignant scarlatina as early as the fifth or sixth day. In this form the eruption does not fade entirely before the tenth or twelfth day, and all the stages are more protracted than in the first variety. Sometimes a persistently *high temperature* may attend this form, depending upon

nephritis and other complications, which are rare in scarlatina simplex. Desquamation is a marked feature of this form, the entire palm of the hand or sole of the foot sometimes peeling away in a single patch.

Scarlatina maligna, like the other forms, usually occurs in epidemics. It is markedly fatal, from twenty to fifty per cent of the cases terminating fatally, in different epidemics. Two varieties of scarlatina maligna may be described, the *nervous* and the *sloughing*. In the *nervous* variety the cerebro-spinal system seems profoundly involved from the very beginning, the patient passing into the most violent symptoms at once. The chill may not be remarkable, but the vomiting is extreme from the commencement, often attended by purging. The temperature rises to 107° or 108° F., early, convulsions, violent delirium, or coma, quickly following. The onset of the disease seems overwhelming, and a condition of collapse is liable to be reached by the fifth or sixth day, the eruption perhaps never appearing. By this time, the breathing is rapid and shallow, the pulse fluttering, the countenance haggard, and the skin clammy, as the patient lies in a state of coma. Dissolution rapidly follows. In another form, the *throat* mischief is excessively developed by the fifth or sixth day, the fauces becoming remarkably swollen and tender, and deglutition very difficult and painful. The throat is found deep red or dark purple in color, and dotted with patches of ashy-gray exudation with blackened edges. The lymphatic glands at the angles of the jaw and the connective tissues around them are swollen and inflamed, marking the sites of subsequent sloughing. The face is livid and haggard; the pulse is quick, feeble, and fluttering; sordes appear upon the teeth and lips; the tongue is dry, brown, and cracked; the breath is offensive and putrid; an ichorous discharge exudes from the nostrils, and soon rapid and wide destruction of tissue occurs, involving the soft parts about the fauces, and even perforating the skin from the inside. Œdema of the glottis may now occasion suffocation, hemorrhage from destruction of important vessels may exanguinate the patient, or pyæmia may slowly sap the vital forces. Such cases are almost certain to terminate fatally.

Scarlatina is a treacherous disease, and the mildest case, therefore, is often fraught with danger. There are always liable to arise *complications*, which may bring about a fatal termination. In some apparently mild epidemics, where the scarlatinal attacks seem only that of the simple form, nephritis will follow or attend the desquamative process, and anasarca and albuminuria prove fatal. Or inflammation of the middle ear may arise during the course of an appar-

ently mild attack of the disease, and terminate in symptoms of meningitis. Other serious complications, such as typhoid symptoms, may arise, where, in the beginning, and even as late as the termination of the eruptive stage, the disease is apparently of the mildest character. I will briefly consider a few of the more common complications and sequelæ of this affection.

Complications and Sequelæ.—Probably the most frequent complication is that which arises from *kidney mischief*. The first symptom which will attract attention here usually, will be anasarca. The child may appear to be doing well and convalescing properly, when suddenly it is observed that a marked dropsical condition has arisen. This is during or immediately following the desquamative period. The disposition of many is to ascribe this to sudden chilling of the surface when the skin is poorly protected by epidermis; and this may have a bearing, but it seems that certain epidemics are attended by a predisposition to such a complication. As such cases have been almost unknown in my practice, I am disposed to ascribe them to too heroic treatment in the beginning; to the use of too active diaphoretics or diuretics—more especially diuretics—which set up an irritation which the natural tendency of the disease carries forward to an actual nephritis. In all cases of scarlatina, it is best to avoid unnecessary stimulation of the kidneys; and it is difficult for me to understand why they should be stimulated at all. Constitutional symptoms, such as headache, vomiting, restlessness, and a return of pyrexia, the temperature rising two or three degrees, but the pulse becoming markedly slow and full, now attend. The urine will be found to contain albumen and casts, as well as blood-corpuscles. Sometimes hematuria will be present. In two or three days, in favorable cases, the fullness of the tissues will gradually subside, the swelling of the hands, feet, abdomen, and tissues generally, will become less marked, the patient will brighten up from the depression resulting from this condition, the appetite will improve, the urine clear up, and recovery go on uninterruptedly. In unfavorable cases, the pulse becomes more feeble but increased in rapidity, the anasarca increases, the patient passes into a condition of coma or convulsions, and death terminates the case.

Inflammation of the serous membranes is another sequel of scarlatina. Among these, the part most liable to be involved is the endocardium, and fatal endocarditis is liable to be the final result. Pleuritis, peritonitis, synovitis, sometimes going on to suppurative arthritis, but more commonly to the joint symptoms of inflammatory rheumatism, are sequelæ of this disease.

A very serious complication is *diphtheria*, which occasionally

occurs, and is very liable to prove fatal. Unlike the complications already named, which are apt to appear late in the course of the disease, this affection may arise at any time, though it usually occurs during the period of desquamation. The symptoms do not differ from those of the uncomplicated disease, except that characteristic exudation and *marked depression* are noticeable. A fatal course is almost invariably run when this complication arises, the already debilitated condition of the patient offering feeble foundation for successful treatment.

Among other complications which occasionally attend may be named *eye affections*, such as keratitis, retinitis, and complete loss of vision. Also anemia, spinal disease, paralysis of single nerves, deafness, chorea, epilepsy, valvular disease, and chronic albuminuria.

Diagnosis.—The diagnosis of scarlatina ought not to be difficult, yet there is often confusion and mistake in identifying it. There can be little excuse for confounding it with measles, for in that disease the catarrhal symptoms are so prominent, in the majoriry of cases, that they cannot be mistaken, while they are absent in scarlatina, especially in the beginning. The eruption of measles appears first on the face, while that of scarlatina shows first on the neck and breast. The fever of scarlatina persists after the appearance of the eruption, while in measles, it falls. The catarrhal features of measles, however, constitute such a distinctive character that there is hardly a possibility of confounding it with scarlatina.

Confluent *small-pox* may at first resemble scarlatina, so far as the eruption is concerned, though the "shotty" feel of the papules will here be an aid in diagnosis, and the first vesicle will settle the question.

Rubella (roseola) is the disease which is most commonly mistaken for scarlatina, and many physicians seem not to know that such a disease as rubella exists, their diagnosis of all such cases being that of scarlatina. There is a great deal of resemblance between rubella and mild cases of scarlatina (scarlatina simplex), though it would be almost impossible for an epidemic of mild scarlatina to occur without the occasional cropping out of some of the sequelæ, such as anasarca, otitis, etc., while these are almost never known in rubella. The constitutional symptoms of rubella are also out of proportion to those of scarlatina, when the comparative severity of the throat symptoms are considered. In roseola, also, there is much less permanency of the white line left when the finger is drawn over the skin, than in scarlatina. The period of desquamation in roseola is not marked, and only a slightly branny scaling occurs, and seldom ever the large patches which are cast off from

the palms and soles in scarlatina, though such a result sometimes happens.

Cases of malignant scarlatina may occur in which there is no eruption before death, but the presence of a prevailing epidemic will readily point out the character of the disease. It is not probable that there will be any confusion in the diagnosis between diphtheria and this disease, the ashen-gray exudation and marked prostration of diphtheria, without the heat of the skin which marks scarlatina, serving to point out the difference.

Prognosis.—Scarlatina is a treacherous disease, and the prognosis should always be guarded. After a few cases have been seen throughout their course, and the epidemic has been shown to be mild, of course a guardedly favorable prognosis may be rendered, where an epidemic of scarlatina simplex is prevailing, and where the throat affection is slight and constitutional symptoms are mild, the eruption appearing within forty-eight hours from the commencement. The more abrupt the onset, such as vomiting, delirium, etc., the darker the eruption, and the more severe the throat symptoms at an early stage of the disease, the more doubtful the prognosis. Age exercises considerable influence, infants and children up to the age of five years being the most unfavorable subjects. Beyond this age, up to adult life, the prognosis is better. Adults affected with cardiac or renal disease, and pregnant women, are unfavorable subjects. An epidemic may develop some peculiarity, such as malignancy, anasarca, etc., the knowledge of which will have an important bearing upon the prognosis during that season.

Treatment.—*Prophylaxis.*—As this is a highly contagious disease, it is important that patients affected with it, as well as those who have been exposed, should be *strictly secluded* from those not infected. Scarlet-fever patients should be confined to the sick room until desquamation has completely ceased; and as long a time as three weeks should be allowed for this, after the period of desquamation has begun. Unnecessary furniture should be removed from the room, thus leaving as little material to act as *fomites* as possible. The clothing and secretions of the patient should be thoroughly disinfected, as in typhoid fever, and during the period of desquamation, measures should be taken to prevent the dissemination of particles of dry cuticle. These will consist in the use of warm sponge *baths* and fatty *inunction*, or the use of olive oil upon the skin after sponging. *Nurses* and others much about the sick room should not have intercourse with those who have not been exposed, until after the period of desquamation has passed, and their *clothing* should be carefully disinfected before being worn in public. The *apartment*

occupied should be disinfected and aired, with the windows open for several weeks prior to further occupancy. The *funerals* of those dying with this disease should be private, that danger of spreading the infection in this way may be avoided.

Medicines have been recommended to prevent the development of the disease after exposure, or to lessen its severity. Belladonna has long been recommended as one of these, but there is much disagreement as to its virtue. I think it exerts little if any formidable influence in genuine scarlatina. Arsenic has been recommended by some who deny the virtues of belladonna. It should be administered in minute doses of Fowler's solution in such case, and its use should be begun as long as possible beforehand. Echinacea promises more, to ward off the severe forms of the disease, than all other remedies; for, though it may not be considered prophylactic in the strict sense of the word, it fortifies the blood against sepsis, the tissues against phagedena, and the cerebro-spinal centers against acute morbid changes. In malignant scarlatina, all susceptible persons who have been exposed should take three or four doses of this medicine per day, one drop for each year of age being a good rule to follow in dosage.

The *medicinal treatment* of scarlatina cannot be reduced to a routine practice. So many varying conditions confront us in every epidemic, that individualization of cases in treatment will be the only successful plan to pursue. However, a few suggestions may aid the practitioner to meet the various conditions which arise. The principal requisite is a knowledge of the proper application of dynamical therapeutics.

In the *treatment of scarlatina simplex* little is required, except the use of *aconite and phytolacca*, in small doses, frequently repeated, to control the fever and throat irritation, thus guarding against sequelæ and complications, as the disease progresses. During exfoliation and convalescence, the patient should wear flannels next the skin, and the surface should be anointed with lard twice a week, to protect the denuded surface against sudden chilling, thus acting as a safeguard against nephritis and other complications. Bathing must be avoided for several weeks. The diet should be light and assimilable, a liquid diet taxing the throat the least. *Malted milk* here serves a valuable purpose. The dose of aconite will vary from one to five drops in four ounces of water; dose, a teaspoonful every hour, amount depending upon the age. If an active agent like Lloyd's aconite be used, care must be observed that the dose be not so large as to embarrass the vital processes. *Convalescence* is a protracted period in recovery from scarlatina, and too much care cannot be

taken now, as it is the most critical time of scarlatina simplex, and a critical period in all other forms of the disease as well.

The *treatment of scarlatina anginosa* will demand the employment of a wider range of remedies. The febrile action here is so high, and the cutaneous irritation so marked, that aconite does not supply the demand for a pronounced sedative. Where there is not the urgent gastric symptoms, manifested by nausea and vomiting, the most acceptable sedative is *jaborandi*, which lowers the temperature, imparts a cooling influence to the cutaneous surface, and controls the irritation of the fauces, to considerable extent. Sometimes, however—indeed often—we shall be obliged to dispense with this agent, on account of the nausea and vomiting, and depend upon *aconite and rhus tox.* Where jaborandi is admissible, I add from one to two drachms of the specific medicine to four ounces of water, and give a teaspoonful every hour, until the eruption is well out, and until the fever has begun to decline. Where the gastric irritation is marked, nothing will do better, usually, than a combination of minute doses of aconite and rhus, though sometimes a resort to *bismuth, peach-bark infusion, ipecac*, etc., will answer better. However, a very certain indication for *rhus tox.* is the "strawberry tongue," and here we almost universally have it. The *throat symptoms* are always severe here, and will occupy considerable of our attention. Our sedatives will assist some in relieving the tumefaction and pain, but we must prescribe something still more positive. *Phytolacca and echinacea*, in combination, will here be found to serve an excellent purpose. Half a drachm of specific phytolacca and two drachms of specific echinacea in four ounces of water, for a child five years of age, will not be too strong. Of this we may administer a teaspoonful every half hour, in severe cases, until relief follows. Often, much benefit may be obtained by using a *spray* of echinacea, one part to four of water. Where there is dryness, with much burning and pain, it affords considerable relief. Sometimes a better remedy, used as a spray, is a drachm of essence of peppermint to an ounce of water to which has been added ten drops of carbolic acid. To assist the action of the sedative and other treatment in quieting irritation and restlessness, lowering the temperature, etc., the frequent application of warm *alkaline baths* is advisable. Cold water is not so good in scarlet fever, for, while it affords temporary relief, there is some doubt about the advisability of applying cold water where the skin has been subjected to so much debilitating influence. *Local applications* to the throat may or may not amount to much. One thing is certain, however, they afford comfort to patient and friends, in that something is being done in this direction, and

should not be omitted. The throat may be swathed with cloths wrung out of *vinegar and water*, or a solution of *hydrochlorate of ammonium*, half an ounce of the salt to a pint of water. *Fatty inunction* over the surface of the body is an important measure after the stage of desquamation has begun, as it lessens the liability to serious complications, and fortifies the skin against sudden chilling. Where there is extension of the inflammation along the eustachian tube to the middle ear, *pulsatilla* and *piper methysticum* are valuable agents to prescribe—ten or twenty drops of pulsatilla and ten or fifteen drops of piper methysticum to four ounces of water; dose, a teaspoonful every three hours. In persistently elevated temperature, where the ordinary sedative treatment fails, the *salicylate of ammonium* will be found an excellent resort, where the stomach will retain it. During convalescence, a subnormal temperature is a common condition, and will call for *echinacea*, *arseniate of quinia* 3x, *nitroglycerine*, or sometimes, small doses of *sulphate of quinia*.

The *malignant* form of scarlatina presents us with the class of cases pronouncedly demanding *echinacea*, when the nervous phenomena are marked. Where vomiting is persistent, and the medicine cannot be retained in the stomach, an effort should be made to derive its effects by hypodermic means. Subcutaneous injections of the drug may be made, and its effects obtained, even though the stomach will not retain it. Where the tongue is heavily coated from the beginning, a thorough emetic will prepare the way for other treatment and interrupt the force of the disease, in a great measure. Sometimes the hypodermic use of Aulde's nuclein may be of service here. The *phagedenic* form will be much modified by free doses of *echinacea*, administered during an early stage, and continued throughout the course of the disease. *Comatose* conditions may be benefited with small doses of *belladonna*, though *baptisia* or *echinacea* should constitute a large part of the internal treatment in this case. Where extensive *sloughing* occurs, nothing will do better as a local application than *echinacea*, one part to three of water, applied frequently. Burrowing ulcers may be syringed with it, the throat may be gargled with it, and cloths saturated with it may be kept bound over the external surface.

Anasarca will suggest the use of *apocynum*, *convallaria majalis*, or *digitalis*. Febrile conditions will here be met appropriately with *jaborandi*. Sometimes an active hydrogogue cathartic will assist in reducing the swelling, and diverting renal irritation. *Vapor baths* are applicable to this condition, and alone will often succeed in removing the morbid accumulation and relieving the renal obstruction.

Many other sequelæ may arise which will require attention long after the termination of the disease proper, and they therefore do not come under this department.

IX. MEASLES.

Synonyms.—Rubeola; Morbilli.

Definition.—An acute, contagious disease, characterized by an eruption which appears on the fourth day, preceded and accompanied by marked catarrhal symptoms notably affecting the bronchial tubes, and a fever of moderate height.

Etiology.—Measles is a highly contagious disease, and it is not probable that it ever arises spontaneously. The breath is supposed to contain the elements of infection, at least the catarrhal elements from the mucous membrane seem to contain the infectious principle in the most concentrated form, though it has been proven that the blood contains the element of the disease, as its inoculation imparts the infection from the sick to the well. The infection seems to be volatile, as a brief exposure of infected fomites to the air renders them innocuous. Experiments upon measles patients for the object of proving that the breath contains the elements of infection, made by causing affected persons to breathe through glass tubes coated on the inner surface with glycerine, resulted in the discovery, microscopically, of bacteria, which develop to a certain point in their career, in a proper medium, and then disappear. They have also been found in the blood, in the true skin, in sweat glands, and in the lymph spaces. They occur in a variety of shapes: ovoid, spherical, rod-shaped, spindle-shaped, etc. The infection begins with the commencement of catarrhal symptoms, and continues until some time after the rash has faded. A single attack usually confers immunity, though two or more attacks occasionally occur in the same person. It is largely a disease of children, because it is so intensely contagious that children are not liable to escape it, but adults are fully as susceptible as children if not protected by a former attack, and it is much more severe in its effects when attacking adults than children. Endemic in populous centers, it becomes epidemic at intervals.

Pathology.—Autopsies of cases resulting fatally furnish evidence that some complication and not the disease itself, strictly, has been the occasion of the fatal issue. The common febrile changes in the blood, such as loss of fibrin, lack of coagulability, and dark color, with hypostatic congestion of the lungs, hyperæmia of the mucous membranes, liver, and other internal organs, with extravasation into their structures, are present. The skin affords evidence of active alteration, in swelling of the corium, as well as of the

rete Malpighii, from active cell proliferation, this extending along the hair and sweat-gland ducts into the glands. The bacteria already described as existing in the breath are found in the liver, the external layer of the cutis vera, in the sweat glands, in the lungs, and other parts. In most severe cases of measles, capillary bronchitis is a common attendant or complication, and catarrhal pneumonia is commonly associated with it. The severity of measles depends much upon epidemic influences, a cerebro-spinal complication sometimes prevailing, rendering an epidemic extremely fatal. Gastro-intestinal hyperæmia may sometimes be marked, giving rise to gastric and enteric symptoms. Hyperæmia of the conjunctiva is a common condition, and in adults who are severely affected this may involve the Meibomian glands and even the lymphatics associated with them, terminating in lymphangitis and suppuration, as well as suppuration of the Meibomian glands, to be followed by chronic irritation of these structures, with frequent recurrence of swelling and suppuration of the edges of the eyelid, as long-perpetuated sequelæ.

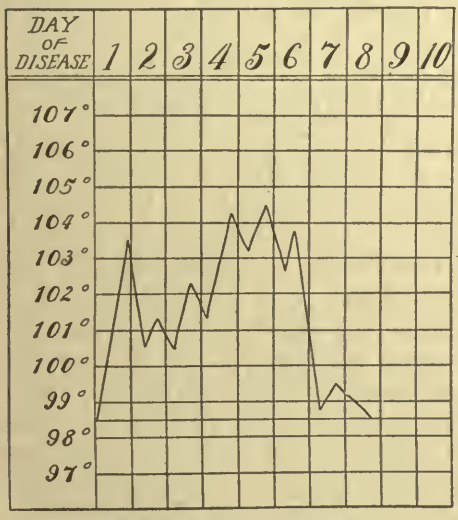
The eruption is papular in character, the papules first appearing on the face. They gradually extend to all parts of the body, appearing last upon the back of the hands. The papules constitute the red patches, and are the result of inflammation of the sebaceous follicles, each representing the center of a reddened semilunar patch. In the center of each patch, in many places, will be found a central hair. The papules may be felt better by the finger than seen, in many cases. If there has been profuse sweating, the epidermis may be raised into small vesicles, or if the inflammation has been very active, there may be extravasations of blood into the skin. Sometimes there is coalescence of the patches in places, especially about the face, and œdematous spots result.

Symptoms.—The average *period of incubation* is estimated by reputable authors as about eight days, though frequently a much longer time elapses after exposure before the disease develops. Some authors give two weeks as the average time. From eight days to two weeks may, then, be regarded as a fair estimate. During this time, the disease remains latent, the subject experiencing no knowledge of its presence.

The first noticeable feature of an *onset* of measles in a child will probably be announced by the appearance of catarrhal symptoms. The patient is observed to be troubled with "snuffles," cough, probably, watery eyes, with photophobia, and he is peevish and fretful, complaining of headache and chilliness. A marked chill is not common, though for a little time now the thermometer may register a

subnormal temperature. The cough is peculiarly harsh and rasping, without moisture. The headache is confined largely to the frontal sinuses, and is dull, aching, and constant. Sometimes the onset is more abrupt, convulsions ushering in the initial symptoms, or the fever being high from the start. At all events, febrile symptoms will become established within twenty-four hours, secretion being arrested, the skin becoming hot and dry, the urine scanty and high colored, there being loss of appetite, thirst, and restlessness.

The *temperature* is not usually excessively high in measles, nor is the pulse so rapid as in scarlatina. The temperature ranges from 100° to 104° F., slight diurnal remissions occurring with gradual rise, until the eruption has reached its height, when it abruptly falls, the temperature reaching normal within thirty-six or forty-eight hours. The *pulse* may run from 100 to 120, and in young children it may run as high as 160, but is not much altered in character, except as regards frequency.



TEMPERATURE CURVE IN MEASLES.

Before the *eruption* appears, an examination of the throat will enable one to detect dark red spots on the soft palate and fauces, which later become diffused over the surface, marking the approach of the cutaneous eruption. The cutaneous eruption appears at the end of the third or the beginning of the fourth day, exceptionally as late as the fifth. It first appears as small spots of raspberry-red color, on the forehead and sides of the face, spreading to the neck, cheeks, breast, and down the body, usually covering the entire surface—lastly invading the back of the hands—in about four days from its first appearance on the forehead. The spots rapidly coalesce into irregular crescentic patches, with intervening spaces of unaffected tissue. About the face these patches often become œdematous.

During the progress of the eruption, the fever becomes aggravated, and the cough is more deep, harsh, and rasping, often being almost continuous. The headache is now severe, the respirations hurried, and wheezing in many instances, and there is marked dread of light, the eyes being sensitive and suffused. There is itching

and burning of the skin, epistaxis is common, and there is enlargement of the superficial lymphatic glands. When the eruption is fully developed, the finger drawn over the surface will leave a white line, which, however, rapidly disappears. The eruption recedes progressively in the course that it pursued while making its appearance, beginning first with the parts where it appeared first and following the line of its development. The marked redness gradually disappears, the redness assuming a yellowish tinge and gradually fading out, until the last sign is a slight staining of the surface.

The *desquamative* stage bears little resemblance to that of scarlatina. Instead of being scaly or branny, the cuticular elements fall off in the form of fine powder, often being unappreciable to the observer, and it is unattended by fever or other constitutional disturbance, the temperature being normal and convalescence established.

Atypical Course.—The regular course of measles may be interrupted by omission of some prominent stage, or marked variation may occur, signally altering the character of the disease. Sometimes the catarrhal symptoms are absent and the eruption appears without any warning. Such cases are styled *morbilli sine catarrho.* Other cases occur in which the eruption is absent, or at least very scanty. In these cases the catarrhal symptoms are marked, and there can be no mistaken diagnosis, as the disease will be prevailing with regular symptoms in other patients. Cases occurring without the eruption are referred to as *morbilli sine exanthemate.* So-called *black measles* occurs in different forms, all presenting evidence of more or less malignancy, and being due to the occurrence of the disease among bad hygienic surroundings. In one form there will be about the same initiatory symptoms, but the temperature will be very high, ranging from 106° to 107°. Restlessness and delirium may now be marked, the tongue will become dry, and the eruption will appear dark from the very beginning, important changes in the blood being so indicated. This may be due partly to epidemic influences, such cases being likely to occur frequently during certain epidemics, while they may not be seen at all during others. In this case also the temperature runs a remarkably high course. In still another class of cases the eruption appears in petechial black spots scattered over the surface, due to a hemorrhagic condition of the patient. In connection with this symptom, hemorrhages from the mouth, nose, stomach, intestinal tract, and kidneys, is liable to occur. These are considered very grave forms of the disease.

Complications and Sequelæ.—Congestion of the *bronchial* mucous membrane is one of the conditions of this disease, but it

may become so aggravated as to constitute a complication. The sonorous râles heard ordinarily over the chest are common to the usual case of measles; but when capillary bronchitis arises, the condition may be considered a complication, as it is by far the most serious element of the disease. There are now small crackling and subcrepitant sounds over the affected area, with absence of the normal respiratory murmur. When lobular pneumonia is present, the symptoms are much the same as these, except that there is dullness on percussion over the affected portion. There is now marked increase in the rise of temperature, the pulse is increased in frequency, the respiration is hurried and difficult, and the countenance is cyanotic in appearance.

The *conjunctival congestion* may also constitute a complication, this sometimes becoming prominent, purulency, with a high grade of inflammatory action attending. Ulceration of the cornea sometimes occurs, followed by rapid destruction of vision.

Stomatitis sometimes occurs as a complication, the mouth being swollen, hot, and dry, and often extensively ulcerated, though this is most apt to be the case where the patient has been mercurialized. Here the fauces and pharynx may be involved, and the trouble may expose the glottis, causing œdema and suffocation. Sometimes it, as well as the other complications, may be peculiar to some certain epidemic, being present in almost every case, to greater or less extent.

Extension of the inflammation of the pharynx into the eustachian tubes, and from there into the *middle ear* may occur, giving rise to suppuration and perforation of the tympanum, and even suppuration in the mastoid cells, to be followed by a long train of unpleasant and serious symptoms.

Cerebro-spinal meningitis occasionally occurs as a complication of an epidemic of measles, the disease presenting many of the symptoms of cerebro-spinal fever. When this occurs, the disease becomes protracted and stubborn. The rash may fade out, the bronchial symptoms persist, and continue until the patient is worn out and exhausted. Or, convulsions may set in and terminate the case speedily.

Diagnosis.—With the dry, harsh cough and other catarrhal symptoms of measles combined with the eruption, the disease can hardly be mistaken for any other of the exanthemata. In no other affection is the persistent cough so noticeable. If, however, it should happen that a severe pulmonary complication should attend rubella or scarlatina, the watery eyes and nasal symptoms would hardly be counterfeited. In children, the eruption of typhus fever might resemble that of measles, but in typhus the rash does not

appear on the face, while here is the first place it appears in measles. Nor is the eruption accompanied by catarrhal symptoms in typhus, while the delirium of that disease is absent in measles, unless it be the typhoid form. Then, the history of the case will go far toward settling any matter of dispute of this nature. The absence of severe cough and other catarrhal symptoms will be a diagnostic point where there might be a mistaken identity between this disease and rubella. Early enlargement of the posterior cervical glands in rubella would assist in the discrimination, as well as the marked congestion of the fauces, which is not very prominent in measles, even though the exanthem is early spread over the soft palate.

It is hardly possible to confound measles and scarlatina, as the catarrhal symptoms are so much more marked in the former, while the inflammation of the fauces in the latter disease is of a pronounced character.

Prognosis.—The prognosis of this disease will vary much, according to the circumstances attending. When the patient is placed under favorable circumstances for good nursing and this is supplied, hardly any uncomplicated case ought to terminate fatally. A warm room, where the air can be kept at an even and somewhat elevated temperature, is essential to a favorable recovery from the bronchial irritation. The poor, then, who are badly housed, and whose houses are but indifferently warmed, and those who are unavoidably exposed to cold air, such as soldiers, are not favorable subjects during winter weather, as serious pulmonary complications are almost certain to arise, which render the prognosis unfavorable. The age of the patient also exerts an important influence upon the question of prognosis, children recovering with much less liability to serious complication than adults. Black measles, either the hemorrhagic, ulcerative, or typhoid form, presents us with grave difficulties, and the prognosis should be extremely guarded. Measles occurring during pregnancy is liable to prove fatal to the fœtus, absorption being the probable result, though the mother may not suffer severe effects. Marked disturbance of the menstrual function is liable to follow a severe attack of measles in the adult female. It has been said that when a pregnant woman passes through an attack of measles without aborting, the child is as well protected against future attacks as though affected after birth. Capillary bronchitis, pneumonia, croupous laryngitis, diphtheria, and cerebro-spinal meningitis, are all serious complications, and the presence of either of them might be considered as reason for a guarded prognosis.

Treatment.—The *preventive treatment* consists of strict quar-

antine against the disease, confining those who are affected to separate quarters, and isolating the nurses and attendants. Excretions, and all clothing worn by the patient and nurses, should be thoroughly disinfected, as in typhoid fever, and the quarantine should continue until the period of desquamation has ended. It is hardly desirable to quarantine all epidemics of measles, as children suffer less from the disease than adults, except during infantile age, and, as the contagion almost always exhausts every community of unprotected subjects, an escape during childhood only destines the adult to a more severe attack during maturity. However, as some epidemics are attended by malignant symptoms in almost every case, avoidance of the disease is always commendable at such times.

The *medicinal treatment* of uncomplicated measles is simple, though the practitioner should be on the alert for complications, that their severity may be modified early. To control the congestion of the mucous membrane to some extent, control the cough and encourage early departure of the cough and coryza, the use of small doses of *jaborandi* and *asclepias*, in combination, serves an excellent purpose. Add a drachm of each to half a glass of water, and give a teaspoonful every hour. This will modify the cough, assuage the fever, quiet restlessness, and impart a sensation of coolness and comfort to the skin, as well as lessen the severity of all the congestive symptoms. Alkaline baths to the skin are grateful, though they should be warm. The room should be darkened so as to prevent the bright light from irritating the eyes, and when these organs are much irritated, cold compresses are of service to assist in preventing later complications and sequelæ of this character. The temperature of the room should be warm and equable throughout the course of the disease, as cool air is aggravating to the cough.

Sometimes the patient will object very much to jaborandi, as well as asclepias, and something less objectionable may be required. *Aconite* and *rhus tox.* afford satisfaction in the majority of cases, and this combination is especially to be recommended where there is marked restlessness, with a tendency to delirium at night. Add five drops or less (according to the age of the child) of aconite, and ten or fifteen of rhus (or less in very young children) to half a glass of water, and give a teaspoonful every hour. This is essentially the prescription where there is gastro-intestinal irritation, with nausea, vomiting, or diarrhœa.

The *cough* sometimes proves a stubborn symptom, and demands especial attention. One of the most successful remedies for the cough of measles is *drosera*, which may be administered by adding ten or twenty drops to four ounces of water, and giving a teaspoon-

ful every hour. Sometimes this will fail to afford satisfaction, and we will need to resort to *sticta*, *sanguinaria*, or *ipecac*. *Echinacea* will sometimes cut short a cough of this character after more approved drugs have failed. When the cough arises from catarrh of the smaller tubes—capillary bronchitis, evidenced by stuffy, suffocative cough, purple, cyanotic countenance, and dyspnœa—*tartar emetic*, 2x or 3x (3x for children), will be the proper remedy. Two or three grains may be administered at a dose, repeated every two hours until the special symptoms demanding the remedy disappear. Where chronic catarrhal symptoms persist, three grains of *calcarea carb.*, every three or four hours, may be administered to complete the cure.

Malignant measles demands remedies to correct depraved states of the blood, in the majority of instances. Specific indications will often point out the proper agent to correct the basic lesion and place the system in such a condition that the ordinary remedies are successful in these cases. Where there is excessive acidity of the stomach, as manifested by the broad, flabby tongue, with pasty white coating, *sulphite of sodium* will be the proper corrective. It may be given in from one- to three-grain doses every two or three hours, until the marked indications are removed. The brown coating on the tongue will suggest *sulphurous acid*, and this should be given a leading place in the treatment, until the marked indication for the remedy has vanished. Ten or twenty drops of a reliable preparation, well diluted with water, should be administered at a dose, every three or four hours. During the course of the disease, the clean, slick, dark-red tongue (beefsteak tongue) may be developed, calling for appropriate doses of *hydrochloric acid*. In many of these conditions the patient will be very sick, some peculiar unpleasant feature or symptom being more than ordinarily prominent. Typhoid symptoms are almost certain to be present, a strong tendency to delirium or coma attending, and the temperature running high in comparison with that of ordinary cases. Where there is a tendency to coma, with cold extremities, the sedative in these cases may be replaced with minute doses of *belladonna*, two drops of the specific medicine to four ounces of water, combined with minute doses of aconite; dose, a teaspoonful every hour. In the majority of these cases, however, *echinacea* will answer a better purpose, its corrective influence on the depraved condition of the blood being better, while it is as positive a remedy to improve the capillary circulation as belladonna. In the petechial form, as well as that attended by ulceration of the mouth, echinacea will be found an excellent resort to correct the depravity of the blood and prevent

phagedenic processes. Our older therapeutists would have proposed baptisia here, but this was before the profession was acquainted with the remarkable properties of echinacea.

In epidemics where cerebro-spinal disturbances are marked, we will find *echinacea* our best resort, it being the best remedy we possess for cerebro-spinal fever, or for its complications. A drop for each year of age up to fifteen will be as satisfactory a plan of dosage as any other, for this remedy. It may be repeated every two hours, in urgent cases.

A markedly high temperature may sometimes attend, and sometimes we find this present where the extremities are cold and the circulation in the superficial capillaries feeble. Where the remedies already suggested fail to produce a sedative effect, I would suggest the use of *salicylate of ammonium*, in appropriate doses.

Convulsions occurring during the onset of this disease will not require special treatment, aconite and rhus, as directed for ordinary sedative purposes, being the proper remedies here. If called during the convulsive action, the physician may direct the child to be put in a warm bath, or wrapped in a flannel blanket wrung out of warm water. As soon as the action of the sedative is established, the signs of convulsions disappear.

Warm alkaline baths are sometimes of service where the eruption is attended by severe burning and itching, and in black measles where there is feeble capillary circulation, or where there has been a retrocession of the rash, as well as where the rash is tardy in making its appearance and the patient seems to be suffering on that account, a sponge bath of aqua ammonia diluted in water (an ounce to a quart of water) will often assist.

Where pulmonary symptoms are pronounced, a pack of cloths wrung out of tepid water, applied to the chest, will be found an excellent auxiliary to the properly selected remedy, frequently affording speedy relief to urgent symptoms, such as dyspnœa, cough, etc.

The *diet* during measles should be mild and unstimulating in character, but nutritious. Plain milk, lime water being added for young children, or what is better, malted milk, will furnish all the nourishment needed during the active period of the disease. Later, during the desquamative stage, solid food may be taken, moderately at first. Cold water may be allowed freely during all stages of this disease, as well as during all fevers, provided there is no gastric irritation present, to contraindicate its frequent use. Ice water, however, is too cold.

During the stage of desquamation and immediately afterward, the skin is poorly protected against draughts of air and sudden

changes, and should be well clothed, as there is almost as much danger to the pulmonary organs as to the kidneys during the late stage of scarlatina. Inunction of the skin with lard or olive oil is a commendable measure where there is the least possibility that the patient may become chilled. During winter, spring, and autumn, in the Eastern States, this measure is an important one. Flannels should be worn invariably, for several weeks after convalescence.

X. RUBELLA.

Synonyms.—Rötheln; German Measles: Epidemic Roseola.

Definition.—A specific, mildly contagious, eruptive disease, resembling measles many times in its eruption at others scarlatina, and resembling scarlatina in the accompaniment of sore throat, but lacking the cough and other catarrhal symptoms that characterize measles, and the high temperature and sequelæ that usually attend scarlatina.

Etiology.—Rubella occurs epidemically, and is feebly contagious. The contagious character of the disease, however, is not so marked as that of scarlatina and measles, several instances coming under my observation where one in a family of children has been affected while the others escaped, and there were many other isolated cases in the same neighborhood; though usually the majority of children in families are affected, when it is once introduced. It seems that the intensity of the contagious principle differs during different epidemics, the disease sometimes manifesting marked contagiousness, and again seeming to be but slightly contagious, if at all. This is probably the reason why there is such a difference of opinion among medical authors on the subject, some claiming that it is eminently contagious, while others of fully as much reliability aver that it is not contagious at all. In some epidemics, children are the only subjects affected, while in others, adults are commonly attacked. In such cases, adults suffer fully as much, if not more, than children. It is asserted that a single attack affords protection from subsequent ones of the same disease, though it is certain that it affords no immunity from measles or scarlatina.

Pathology.—The most marked pathological changes occur in the skin, throat, and cervical glands, though these are not of radical character. The skin-changes consist of irregular hyperæmic blotches, which vary in size from a pin's head to a fourth of an inch in diameter. They are slightly elevated, but disappear under pressure, and do not impart the hardened feel to the touch that is observed in the early stage of measles, indicating less plastic exudation, and less

inflammatory action. The eruption appears upon all parts of the body, and the patches are round—not crescentric, like those of measles. There is no exudation of serous or lymphoid material into the rete Malpighii as in scarlatina, nor inflammation of the sebaceous follicles, as in measles. The throat is congested, sometimes markedly so, and the cervical lymphatics, especially the posterior cervical, are swollen and tender, even early in the course of the disease. Slight powdery desquamation from the skin occurs, but seldom in the form of flakes, as occurs in scarlatina. The important congestion of internal organs that marks the more severe forms of eruptive fevers, is not present to any considerable extent in this.

Symptoms.—The stage of *incubation* is said to be about two weeks in length.

The stage of *invasion* is hardly noticeable in many cases, though there may be a distinct chill, and even convulsions, during the attack. The *eruption* occurs within twenty-four hours after the invasion, though many cases manifest no unpleasant symptoms until the appearance of the eruption, the child being at play when it is first noticed. It appears first on the face, and spreads over the entire body within two or three days, disappearing within twelve or twenty-four hours after its appearance, a progressive subsidence following the march of its appearance. The rash varies in color from the scarlet appearance of that of scarlatina to the raspberry color of measles. Often it is but faintly marked, though the more pronounced the rash the more severe the other symptoms. The rash is often distinctly separated into little spots or patches, though again it may be evenly spread, as in scarlet rash. Desquamation occurs as a powdery exfoliation, the superficial parts of the epidermis only being involved, serious disturbance of the cuticle of the palms and soles not being noticeable, as in scarlatina. The *pulse* is increased in frequency, being, in some cases, small and wiry. The *temperature* is slightly elevated, one or two degrees being the average, though in severe cases it may reach 103° F.

The *throat* symptoms are sometimes apparently quite severe, the tumefaction and difficulty of swallowing being marked and troublesome, though the irritation is superficial, as manifested by absence of sloughing or destructive action later. Sometimes the follicles of the tonsils are involved, and white patches of exudative material appear about their orifices. The redness, however, does not spread over the palate, as in scarlatina. The muscles of the neck are often stiff and painful, and the muscles of the body generally are sore, the patient complaining of a bruised feeling. Muscular rheumatism may attend severe cases. The cervical lymphatics will be found swollen

and tender early in the course of many cases, especially the occipital lymphatics. Most cases are so mild that the patient considers it a hardship to remain in bed, and, if allowed, he will be up and around before the appearance of the eruption.

The *tongue* is coated with a thin white coating, early, through which dark red points appear, and later the organ may appear slick and dark red in color. The organ is usually pointed and reddened at the tip, though there is rarely nausea or vomiting.

Complications and sequelæ are almost unknown in this disease, and although these are mentioned by authors as occasionally occurring (anasarca, for instance), it seems that such can only be the case where a very bad plan of treatment has been pursued.

Diagnosis.—Probably there is no other disease known in which physicians make so many blunders in diagnosis as in this. The *blunder*, however, is usually on the safe side—for their credit—as they commonly diagnose it as scarlatina. The great number of cases of scarlatina which occur around us with no mortality would be surprising, were it not for the fact that the physicians who manage them are the most arrant blunderers in therapeutics possible, in most instances, and really sometimes find it difficult to pull a simple case of rubella through successfully. About as positive a diagnostic point as can be scored then is that if no mortality or sequelæ attend we are having an epidemic of rubella. Mark the low temperature, the absence of violent gastro-intestinal symptoms, of delirium, and destructive action in the throat, which attends scarlatina. Mark also the absence of pronounced catarrhal symptoms, the absence of the rough, deep cough, which announces measles to the whole household. Mark, also, the brief stay of the rash, and the small amount of pyrexia during its presence, and you will find enough distinctive features to determine a case of rubella.

Prognosis.—Without the most absurd and irrational treatment, and the worst nursing imaginable, and without some unexpected and unwarrantable complication, the prognosis is always favorable.

Treatment.—A combination of *aconite*, *rhus tox.*, and *phytolacca*, from one to five drops of aconite, fifteen to twenty of rhus, and ten to thirty of phytolacca, in four ounces of water, dose, a teaspoonful every hour, will represent the proper routine prescription, this covering the usual indications. There may be cases in which muscular pain will be prominent, demanding the judicious use of *acetanilide*, *cimicifuga*, or *rhamnus californica*. Itching and burning of the skin may suggest the local use of the *resorcin lotion* recommended for a similar purpose under the treatment of chicken-pox. Finally, fatty inunction, during desquamation, is to be commended.

XI. MUMPS.

Synonyms.—Epidemic Parotitis; Specific Parotitis.

Definition.—Mumps is an acute, contagious inflammation of one or both parotid glands, attended with fever, and usually resulting in resolution, with a tendency to metastasis to the testes in the male, and to the ovaries or mammary glands in the female.

Non-specific or metastatic parotitis may occur as a secondary symptom in certain infective diseases, such as typhoid fever, pyæmia, diphtheria, measles, etc., and usually terminates in abscess. In the idiopathic variety, this, as a rule, does not occur.

Stephen Paget has collected a large number of cases in which injury or disease of the abdominal or pelvic organs, unattended by septic processes, was followed by an idiopathic, non-specific parotitis.

Etiology.—Mumps, like the eruptive fevers, is propagated by contagium, and, like them, one visitation usually confers immunity from subsequent attacks. However, a person having "single mumps" is liable to a later invasion—of the other gland.

Pasteur claimed to have discovered the "bacillus parotidis," but attempts at the inoculation of animals with it have failed, and the nature of the virus is therefore still an open question.

Mumps rarely occurs sporadically. On the coasts of France, Holland, England, and some localities in this country, it is said to be epidemic. Isolated cases are occasionally met with, but it usually occurs in the epidemic form. Mumps is a disease of childhood, the period when the system is most liable to its invasion being between the second year and puberty. Persons who have escaped parotitis in childhood are not necessarily exempt from its influence; in fact, during some epidemics, adults are chiefly affected. Females are not so liable to contract the disease as males. The immunity of infants is attributed to the slight development of the parotids, and the narrowness of Steno's duct. The humidity of the atmosphere undoubtedly assists in the propagation of this disease, the autumn and spring months being the period when it is most frequently met.

Pathology.—The catarrhal inflammation commences primarily in the ducts, and spreads rapidly to the glandular structure. There is at first an intense hyperæmia, resulting in serous exudation and tumefaction. The acini are œdematous, and there is inflammatory, serous, and cellular infiltration of inter-alveolar fibrous structure, the surrounding connective tissue and adjacent parts being more or less involved. The inflammation terminates by resolution, fibrous induration, or suppuration, usually the first. Occasionally, parotitis results in atrophy of the gland.

Symptoms.—The period of *incubation* lasts from one to three weeks, during which there is little premonitory disturbance, though the swelling of the parotids is often preceded by prodromata, the patient complaining of anorexia, nausea, pains in the head and back, and constipation. There is a *chill* (or chilly sensations), followed by fever, quick pulse, scanty urine, and a dry skin. The patient complains of *stiffness* and *tension* in the parotids, and tumefaction, usually beginning on the left side. The usual phenomena of inflammation—heat, pain, tenderness, and swelling—are present. The *pain*, while unpleasant, cannot be termed severe. It is increased by swallowing, speaking, or pressure. The *swelling* extends in all directions, and we have a general œdema of the affected side. The *lower jaw* is greatly restricted in movement, and mastication and enunciation are difficult, and, at times, impossible. Salivation is an occasional symptom. When stomatitis and ptyalism develop, there is considerable fetor. Occasionally, the submaxillary and sublingual glands participate in the inflammation, and cases have been reported where the parotids were unaffected, the swelling being confined to the smaller salivary glands. The lymphatic glands in the immediate vicinity are usually swollen.

In general, the fever is not very high; in fact, some cases run an apyrexial course, the main discomfort arising from the tension over the parotids, and the immobility of the lower jaw.

In from three to four days the disease has fully developed. About the seventh or eighth day resolution and subsidence of the swelling begin, or, as is often the case, the other gland becomes involved. While the above is the usual course, some cases present serious features, there being hyperpyrexia, intense pain, delirium, and great vital impairment.

Complications and Sequelæ.—Where there has been high fever and marked nervous symptoms, *delirium* and even maniacal attacks are noted, or, in severe cases, meningitis, hemiplegia, and coma. Cerebral congestion may result from pressure on the jugular vein. Visual affections are more infrequent, amblyopia being the most serious. Acute albuminuria, gastro-intestinal disturbances, and arthritis, have been noted. Impairment of hearing, persisting, in some cases, is an occasional unpleasant sequela.

The most frequent complication, however, is *orchitis*. The testicular inflammation develops oftenest after the subsidence of the swelling in the parotid, and is largely dependent upon the character of the epidemic—though too early exercise on the feet may be provocative of it under other circumstances, though usually the severity of the original disease has little to do with this condition. Bilateral

orchitis is rare, although at times the second testicle is in turn attacked. It is seldom noticed before puberty. In an epidemic where 495 soldiers were attacked with mumps, Granier found 115 cases of orchitis. The inflammation does not run any regular course, although it seldom extends over a week.

In the female, ovaritis occasionally develops, and, infrequently, mastitis or vulvo-vaginitis.

Suppuration of the parotid seldom occurs. Where pus forms, there is marked constitutional disturbance, and the pain is severe, as the gland is provided with a strong capsule and the surrounding fascia is deep and firm. As a rule, the pus opens into the auditory meatus, but it may pass along the sheath of the carotid to the skull, burrow its way behind the pharynx into the maxillary joint, or downward into the thorax. It is, therefore, a serious complication.

Treatment.—Mumps is a self-limiting disease, and, in the majority of cases, there is not much call for medicine. Externally, a layer of cotton wadding covered with oil silk is sufficient, although some prefer inunctions of oil or a hot or cold compress. When the pain is severe, a lead and opium lotion gives considerable relief. *Phytolacca* is extolled by some. For the fever, *aconite* is indicated: ℞ Specific aconite gtt. v, specific phytolacca ʒss, aqua q. s. ʒiv. M. S., ʒi every hour or two. At the beginning, there is frequently a deficiency of saliva, and specific jaborandi (ʒii–ʒiii) can be added.

Ptyalism is sometimes present during the later stages, and calls for *belladonna*, or the fractional dose of *jaborandi* or *iris*.

It is seldom that there will be a call for any sedative except aconite, but occasionally there will be an irritation of the nervous system and determination of blood to the brain, calling for the exhibition of *gelsemium* (ʒss–ʒiv), or the sharp stroke of the pulse and nervous erethism, with or without the tongue symptoms, which would indicate *rhus tox.* (gtt. x to xx–ʒiv).

Abscess is a rare complication and must be met promptly by surgical treatment, in order to prevent the danger consequent on the burrowing of pus. Most cases are likely to break in the ear if not interfered with, but the lance should nevertheless be used early. Sometimes it is necessary to make a careful dissection, where the pus is deep. Where we suspect a possible breaking down of the glandular structure, *calcium sulphide* is to be given in small doses with a reasonable expectation of its aborting the abscess.

Orchitis is treated by *rest* and *support* of the testes. Strapping is not often called for. The following lotion is about as good as anything to apply locally: ℞ Plumbi acet. ʒi, tinct. opii ʒi, tinct. aconite ʒss, aqua q. s. ʒvi. M. S., Lotion.

In other cases, a lotion consisting of equal parts of echinacea, phytolacca, and belladonna (green plant tincture or specific medicine), will serve a better purpose. This may be kept applied constantly, with moistened cloths. Internally, we administer *phytolacca* or *pulsatilla*, with our sedative.

XII. WHOOPING-COUGH.

Synonyms.—Pertussis; Tussis Convulsiva.

Definition.—An acute, infectious disease, characterized by the gradual development of a spasmodic cough of peculiar character, signalized by a series of explosive expiratory efforts followed by a long-drawn inspiration attended by a peculiar crowing sound, the "whoop," the cough being preceded by symptoms of a common cold, and followed by a period of gradual subsidence.

Historical Note.—Whooping-cough was described by the ancient Greeks as *bex theroides*. Old writers mentioned it as *tussis convulsiva*. Cullen wrote of it under the name *whooping-cough*, and described it clearly. Considerable discussion was engaged in during past years as to the character of the disease, some claiming that it arose from irritation of the pneumogastric nerve, and others that it was caused by enlargement of the tracheo-bronchial glands. Linnæus foreshadowed the modern microörganism-theory of the etiology of the disease when he ascribed it to an insect. Later, Poulet, Letzerich, and Binns suggested the fungoid nature of pertussis.

Etiology.—The present knowledge of other infectious diseases renders it most probable that this depends upon a specific microörganism, which operates upon some portion of the respiratory mucous membrane. This has not yet been demonstrated to the satisfaction of microscopists, however, though several announcements in the affirmative have been made. Thus, in 1867, Poulet found in the sputa of pertussic patients minute bodies which he termed infusoria, and Letzerich produced the disease in animals by inoculating the trachea with the sputa of affected human subjects, while he asserted that he found a fungus in the secretions of the respiratory passages. Buhl, Oertel, and Hüter also found them. The contagium is given off in the breath and sputa of affected individuals, and probably in emanations of the body as well, as the disease undoubtedly permeates the blood. Children are most liable to the disease, and it usually occurs epidemically, though it may appear endemically. Adults occasionally suffer from it. The mortality among the children of colored races is stated by some authors to be twice as great as that among the white population. Clothing and rooms may be infected so as to convey the disease, in the absence of an affected subject,

though doubtless the common means of infection is by direct contagion. The most common period of life subject to it is that before the third year, though it sometimes occurs during extreme old age. Where the seasons are marked, spring and autumn seem to favor its appearance, and it is supposed in some quarters to be influenced by measles, it often appearing quickly after an epidemic of that disease. One attack usually protects from a second. The infection is believed to persist for five or six weeks after the "whooping" period has passed off, the patient being capable of communicating it during that time.

Pathology.—The most marked changes are found about the respiratory organs. There is catarrh of the air passages—hypersecretion of the mucous membrane of the glottis, larynx, trachea, bronchi and their ramifications—with congestion and hyperplasia. Emphysema is a common condition, as well as pulmonary collapse—in fatal cases. Capillary bronchitis and pneumonia frequently occur, leaving their traces in the post-mortem appearances. There is intestinal irritation, evidenced by petechial extravasations upon the gastric mucous membrane and wall of the small intestine; and the liver and spleen may be enlarged and fatty. Hemorrhage into the subdural space sometimes occurs, and more frequently there are points of extravasation in the brain and spinal cord. There is often an ulcer under the tongue, by the frænum linguæ, in severe cases, due to forcible protrusion of the tongue against the lower incisors during the paroxysms of coughing. The bronchial and tracheal glands are usually enlarged.

Symptoms.—After an *incubation* of from seven to ten days, three stages develop, viz., the catarrhal, paroxysmal, and stage of decline.

The *catarrhal stage* resembles a common cold in its characteristics, there being snuffling of the nose as in coryza, cough, slight feverishness, peevishness, and restlessness at night. The physician may now be requested to administer a remedy for the "cold," and upon doing so he will find that the prescription fails, and he may be applied to for a more successful treatment a second time before it will occur to him that there must be something more than a common cold that will resist well-proven remedies for such a simple complaint. The cough is dry at first, but sooner or later becomes moist, the secretion being a tenacious, viscid, transparent mucus. Paroxysmal symptoms gradually appear, and the cough increases in severity, the secretion being more abundant, the respirations shallow, and the pulse rapid. The duration of this stage varies from three days to three weeks, though it usually lasts about ten days.

The *spasmodic* stage is announced by a paroxysm terminating in

a pronounced whoop, which settles the question of the nature of the disease, and at the same time ushers in a period of severe suffering for the patient, unless the affection be modified by appropriate treatment. The paroxysms of coughing soon become peculiar and distressing. A whistling inspiration, followed by a succession of short, sharp, expiratroy explosions, announces the paroxysm, the expiratory explosions continuing without inspiration until the patient grows cyanotic and exhausted, and seems to have lost the power to fill the lungs or stand upon the feet, the parent or nurse finding it necessary to support the child, which is completely relaxed and helpless in the throes of the paroxysm. The face presents marked evidence of increasing venous stasis, becoming more and more cyanotic, while the eyes bulge out, the lips and cheeks become swollen, the jugulars standing out like blue cords, and the face and limbs being covered with perspiration. The glottis, which is now in a condition of spasmodic closure, finally opens partially, to permit the patient to draw a long, laborious inspiration, which enters the glottis with a sharp, crowing sound—the whoop. Vomiting is now liable to occur, the gagging serving to dislodge accumulated mucus. The child is much prostrated during the paroxysm, and the lower sphincters may be so relaxed that involuntary evacuations occur. If the patient is delicate, it may now fall into an exhausted sleep, or, as in most cases, it may soon recover and go about its play; but it is terrified after a time by the approach of another paroxysm (which furnishes something of a premonition), and may run to its mother or nurse to cling to her for protection and aid. From six to forty or more of these paroxysms may occur in twenty-four hours.

During the paroxysm, the thorax is dull on percussion during expiration, owing to the contraction of the muscles, and remarkably resonant on inspiration, the respiratory murmur being almost indistinct on inspiration on account of the small amount of air admitted through the chink at the time. Between the paroxysms, the respiratory sounds are numerous and variable. There may be sonorous, sibilant, and moist and dry crepitant sounds in the same patient, predominance depending upon the amount and character of pulmonary complication that may have arisen. Bronchitis is often a complication, and where the small tubes are affected, they are liable to become blocked (capillary bronchitis) and occasion cyanotic symptoms and prostration, very much complicating the case.

During the violence of the paroxysms, numerous *accidents* are liable to occur; the pulmonary alveoli may become ruptured and permit of inflation of the cellular tissues of the lung with air (emphysema), which may occasion serious results by permanently

infiltrating the part and interfering with normal function. Rupture of cerebral vessels with apoplexy may occur, or excessive strain to the abdominal muscles may result in hernia or prolapsus ani.

Vomiting after the paroxysms is a common symptom, and this may amount to gastric irritability, with habitual vomiting of food, tending to inanition and marasmus. In most cases, however, the vomiting is confined to efforts to expel the tenacious mucus which accumulates in the throat during the paroxysms, and which is removed with the greatest difficulty, the assistance of the nurse's finger often being required to dislodge it.

Conjunctival, *cutaneous*, and *pulmonary extravasations* often occur during the paroxysms, the eyes becoming bloodshot, and the face presenting purple blotches of extravasated blood, as a result of the violent strain during the act of coughing.

The *nervous system* is in a condition of hyperæsthesia in many cases, the patient being excessively peevish and irritable; cerebral congestion, convulsions, and even permanent insanity have arisen during the course of the disease.

After five or six weeks from the beginning, the paroxysms commence to *decline* in severity; the whoop gradually ceases, and the case starts on the road to recovery, though sometimes the paroxysmal stage becomes chronic, and persists for a year or more. In other instances, whenever the patient may contract a cold, the paroxysms return with considerable severity until after the cold has been dissipated. In about nine weeks from the commencement, in ordinary cases, the paroxysms and cough have ceased permanently.

Complications and Sequelæ.—The sequelæ of pertussis occupy a prominent place in its history. The persistent *vomiting* may give rise to gastro-intestinal irritation, followed by marasmus of persistent character, attended by muco-enteritis, from which the patient may rally with the greatest difficulty, and only under the most approved plans of treatment.

Phthisis, if latent in the system, or if the child be exposed to contamination, often runs a rapid course after an attack of whooping-cough; acute general tuberculosis may develop also. *Emphysema* and *pneumo-thorax*, as well as *broncho-pneumonia*, are complications and sequelæ to be expected, on account of the severe strain upon the lungs.

Treatment.—The treatment of pertussis is not usually applied with very much philosophy. Empirical prescribing is commonly resorted to, and this is the best that we can seem to do with our present state of knowledge. Though pathologists may agree that the irritant is a microörganism, its exact location has not yet been

decided, and if it had, its destruction might involve the use of remedies which would necessarily destroy the pulmonary tissues. If ptomaines are generated, there has been little accomplished toward their correction in the treatment thus far employed. Indeed, old school authorities content themselves (and blight the enthusiasm of their followers) by declaring that there is little that can be done for the disease except to meet complications as they arise. A few cases seem to defy treatment, it is true, but others, and the majority, can be so modified that the course of the disease can be shortened, and little danger or trouble arise from it.

Agents which exert the best influence are adapted to the relief of convulsive tendencies arising from irritation of the pneumogastric nerve, and these are equally adapted to spasmodic cough, whether from pertussis or other provocation.

Of the best of these is *drosera*. It will control a large share of the cases of whooping-cough, and soon banish the whoop—though I have used it where its influence was entirely wanting. When this proves to be the case, the best we can do is to try another remedy. Add from ten to twenty drops of a reliable article of the tincture (homeopathic, or specific medicine) to half a tumbler of water, and give a teaspoonful every two or three hours, in severe cases. If the cough has become pretty well established, it may require a week to bring about the desired effect. If, by the end of this time, there is no noticeable improvement in the cough, it will be rational to abandon this remedy and try another.

A remedy which has proven excellent, and which I have found prompt in relieving the severity of the cough, is *magnesium phos.*, 3x trituration. One or two grains of this may be administered every two hours during the day until relief follows, the number of doses then being lessened.

Where inflammation of the small bronchial tubes, with catarrhal secretion (capillary bronchitis), arises, *tartar emetic* 3x trituration, alone or alternated with *calcarea phos.*, 3x, will be found excellent, calcarea phos. being especially demanded where the child is anæmic, and tending toward a condition of marasmus.

Quinine inunction, or the internal use of *arseniate of quinia*, 3x, may be demanded, where the disease prevails in malarious districts; and sometimes polymnia uvedalia or grindelia squarrosa will be proper remedies, on account of splenic hypertrophy and consequent congestion of the portal circulation. Where a condition of marasmus is well developed (the child having been attacked with convulsions and having entered upon a critical state), the tonic treatment with faradism, repeated every second day for several weeks, will materially assist in tiding the patient through.

Coal-tar products have been highly extolled as remedies for the convulsive cough. I have known coal-miners to carry their children into the mines to remain all day, for the purpose of arresting this disease, after medicines have failed; and it is asserted that this is almost certain to succeed. *Antipyrin* is said to be remarkably efficacious in many cases, in doses of from one to three grains. *Phenacetin* possesses a similar reputation, though it is not as reliable as antipyrin. Acetanilid is less objectionable than antipyrin.

Castanea vesca has proven a satisfactory remedy, and should not be forgotten where stubborn cases are encountered. Ten drops of a tincture of the green leaves should be administered every three or four hours.

Bromoform, in three-drop doses (administered in a swallow of water), repeated three times daily, is reported as nearly a specific.

Inhalations sometimes prove beneficial, and should be resorted to in such cases as seem to defy other measures. The following may prove of service: ℞ Essence of peppermint gtt. x–xx, carbolic acid gtt. iii–v, distilled water ʒi. M. Allow the patient to inhale from a spray apparatus, every hour. Or, a one per cent solution of resorcin may be used instead.

Children recovering from whooping-cough should be warmly clothed to prevent them from taking cold, and, where recovery seems unduly protracted, a change of climate should be advised whenever practicable.

XIII. EPIDEMIC INFLUENZA.

Synonyms.—Epidemic Catarrh; Catarrhal Fever; Contagious Catarrh; French, La Grippe; German, Blitz Catarrh.

Definition.—Influenza is an acute, infectious, epidemic disease, characterized by fever, great prostration, severe pain in the head, back, and limbs, marked nervous phenomena, and catarrh of the respiratory and gastro-intestinal tract. The catarrh may be limited, or affect all the mucous membranes to the same extent.

Historical Note.—The name is not descriptive of the disease, although, as indicating its epidemic character, it is not inapt. The influence (influenza) of the stars was supposed to be causative and, in the absence of pathological knowledge, the rapid spread of the disease from continent to continent was not unnaturally ascribed to stellar influence.

La grippe has prevailed, at intervals, for several centuries, being first described in 1323. Many of the epidemics are historical, such as those occurring in 1831, 1847, and the late epidemic of 1889–90.

At times, influenza has extended over almost the entire globe.

It has traversed the whole of Europe in the space of forty days, the rapidity with which it travels being one of its remarkable characteristics, this probably suggesting the German name "lightning." It has figured in the expression of national dislike and jealousy, as the French call it the "Italian fever;" the Italians term it the "German disease;" the Germans repudiate this by alluding to it as the "Russian pest;" while the Muscovite passes it along as the "Chinese catarrh." However, the majority of epidemics have originated in Russia.

Etiology.—Of the causative germs of influenza, we as yet know nothing. Meteorological conditions have but little influence in its production, and, although the epidemics usually occur in the winter months, they do not differ in character from those appearing in the spring and autumn. Damp, cold, and foggy weather, which would be a prolific cause of colds, would help to disseminate it by rendering the system more liable to invasion, just as local conditions tending to produce diarrhœa and dysentery would favor the spread of Asiatic cholera.

It usually lasts about six weeks, and is severe in proportion to the extent of its prevalency. No class or age is exempt, although children often escape its influence, probably on account of their not being so liable to exposure.

Some of our later investigators do not believe the disease dependent on bacteria, but ascribe it to an organism of a different character. The discovery of the plasmodium of Laveran may have pioneered the way for the discovery of the peculiar microörganism responsible for the production of the disease. One attack does not confer immunity, and repeated seizures are common.

Pathology.—There are no special or characteristic pathological phenomena, the various lesions depending on the different structures involved. If there are marked gastro-intestinal symptoms, the mucous membrane of the stomach and bowels will be found congested. Except in the rarer cases where there is but little catarrhal inflammation of the respiratory tract, we will find it more or less pathologically changed. The lungs are usually distended and protruded, instead of collapsing, when the thorax is opened. The smaller bronchi are much injected, the mucous membrane, here and in the larger bronchi, being inflamed and covered with mucus. A softening and swelling of the bronchial glands is also noted. When pericarditis has been a complication, we have the usual anatomical changes.

Symptoms.—La grippe manifests itself in all degrees of intensity, its clinical features depending on the structures principally

involved, and the complications that ensue. The disease usually begins without prodromes. There is an initiatory chill, followed by fever of a remittent type, ranging from 101° to 102° F. The pulse is not as rapid as one would expect from the fever present, although, in serious cases, it may run up to 120 per minute. The urine is scanty and high colored. With the fever, there are splitting headache, and pains in the eyes and frontal sinuses. The joints and muscles, especially of the back and lower limbs, are racked with *pain*, of a character almost as excruciating as that noticed in dengue and variola. A *prostration*, far in excess of that to be expected from the symptoms, is manifested early, pathognomonic of la grippe. Profuse sweating is usual, throughout the course of the disease. The *catarrhal* symptoms begin in the upper passages, and there are present coryza, hoarseness, soreness in the pharynx and trachea, and a distressing cough, at first dry, but soon changing its character, as the secretion is increased. As the disease advances, the sputum becomes copious and muco-purulent. There is a constriction of the chest, with difficult breathing, præcordial oppression, and feeble cardiac action, in elderly subjects. The involvement of the gastro-intestinal mucous membrane is evinced by nausea and vomiting. The tongue is coated, and usually moist. There is constipation, which frequently gives place to diarrhœa. In epidemics where the digestive symptoms are marked, dysentery is not uncommon. There may be tenderness over the liver, and a jaundiced condition.

The *complications* met with are pharyngitis, laryngitis, œdema and congestion of the lungs, pneumonia, bronchitis, pleurisy, and subacute gastritis. More rarely we have congestion of the liver, parotitis, pericarditis, and various cutaneous disorders. Pneumonia is the most serious complication. Copeland states that in the epidemic of 1831, of the patients at Hôtel Dieu, over 20 per cent had lobular pneumonia.

Ocular disturbances are among the sequelæ, soreness of the ocular muscles, photophobia, and retinal congestion being most common. Loss of vision may occur from the effects of this disease, through retinal hemorrhage.

Diagnosis.—Some of the cases are liable to be mistaken for "bad cold," but the sudden onset, great prostration, and catarrhal features are usually sufficient to demonstrate a case of influenza. After an epidemic is well under way, no mistake should be possible.

Prognosis.—The prognosis is good in the adult where the constitution is not greatly impaired; but the mortality is sometimes quite high among children and the aged. Organic diseases, such as parenchymatous or intestinal nephritis, emphysema, fatty heart, or

pulmonary troubles, render the prognosis more or less doubtful. The disease is serious according to the severity of the complications.

Treatment.—If we are called early enough, an attempt should be made to abort the disease. If we fail in our object, we can, in a great many cases, modify some of the symptoms, and the patient is no worse off for the attempt.

Diaphoresis should be induced by the alcoholic vapor bath, or such remedies as serpentaria (ʒss–ʒi, p. r. n.), jaborandi (gtt. xx–ʒss, p. r. n.), or the diaphoretic or Dover's powders (gr. v–x, p. r. n.). A hot pediluvium should preface the treatment, and the patient should be well covered and hot drinks used, to assist the diaphoretic action of the drugs. Old school physicians attempt to abort the disease with large doses of quinine.

Where there is no great depression, and in the lighter attacks, *phenacetin* will be a valuable remedy; but in serious cases, all depressing remedies should be avoided. Phenacetin modifies the fever and relieves muscular pain better than slower acting remedies. It is well to add *arseniate of quinia* 3x to it in most cases. Three grains of the former to two of the latter every three hours, will be the usual dose. Our sedatives can be given alternately with the phenacetin. *Aconite* (gtt. 1-6th), *veratrum* (gtt. i), or *gelsemium* (gtt. i–ii), will be indicated for the febrile condition, the former being preferred where there is much gastro-intestinal disturbance. *Jaborandi* (gtt. ii–v) may be added, where the skin is dry. For the muscular pain, *macrotys* (gtt. ss) or *arnica* (gtt. 1-10th) are prescribed, but the phenacetin, jaborandi, and sinapisms (the latter moved from place to place as required), will give better satisfaction.

Milk should be the principal diet for the first few days, until the gastric irritability passes away. For this condition, *rhus tox.* (gtt. 1-3d) can be added to the sedative. It will also help to relieve the coryza and frontal pain. A pack or sinapism over the epigastrium usually gives considerable relief.

In the treatment of the respiratory symptoms, we are guided by the nature and extent of the lesion. Where the cough is rasping and explosive, the trachea and its bifurcations being principally affected, *bryonia* (gtt. 1-3d) will usually give relief. *Inula helenium* has been used successfully in past epidemics. Its effects are limited to the bronchi, and by adding to it *asclepias*, we have a powerful combination. They may be given, aa, gtt. v, in syrup and water, every two hours, or oftener, if required.

In acute cough, with dryness and *tickling*, *rhus tox.* (gtt. 1-3d) may be prescribed. It influences both the circulation and the nerve supply, and overcomes that teasing and tickling which is so annoy-

ing. *Sticta* (gtt. ss–i) is a remedy that is not serviceable in ordinary coughs, but frequently does good work in influenza. We do not use it where there is abundant secretion, but where there is dryness and wheezing, the cough being rasping and persistent.

Stannum 6x will meet that not uncommon condition where there is a sense of exhaustion while speaking, with a tired sensation of the larynx.

Inhalations of *tinct. benzoin co.* (ʒss–ʒi in aqua bul. Oi) are grateful to the patient, and help to relieve the catarrhal condition.

At night, the cough can be controlled with *tinct. serpentaria co.* and *glycerine*, aa, ʒss, in hot, sweetened water.

Complications are to be met as they appear, the main object being to avoid depressants, and keep up the strength of the patient.

XIV. DENGUE FEVER.

Synonyms.—Dengue; African Fever; Dandy Fever. When this disease first appeared in the British West India Islands, it was called the *dandy* fever, from the stiffness and constraint which it gave the limbs and body. The Spaniards of the neighboring islands mistook the term for their word, *dengue* (pronounced dĕng'gā), denoting prudery, which also might be considered as denoting stiffness. Thus the origin of the name. It is also termed "break-bone fever."

Definition.—A specific, infectious disease, peculiar to warm countries, characterized by paroxysms of fever, attended by severe muscular and periosteal pains, with anomalous eruptions.

Etiology.—That this affection depends upon a specific contagium, seems proven by the fact that it has been transported in clothing and other fomites from distant parts, on the occasion of more than one epidemic. Its first appearance in this country dates from the landing of a cargo of slaves from Africa. It occurs epidemically and sporadically, and attacks all classes of people, from infant to aged, rich and poor, in common. While most liable to break out in southern latitudes, it has prevailed in more northerly sections, an epidemic having occurred, according to Loomis, in Philadelphia, in 1780. An extended epidemic occurred in the West Indies, in 1827, and one in our Southern States, in 1880. There is some dispute among observers as to whether the disease is contagious or not, and as to whether one attack provides immunity against others.

Dr. McLoughlin, of Texas, has discovered a microbe, which he presumes to be the active causative agent. The organism is a form of streptococcus. Whether it prove to be the genuine cause of the disease may yet be considered questionable.

Pathology.—The pathology of dengue seems to resemble that of malarial fever in many respects, and it was once believed to be a modified form of that affection. Arthritic changes, similar to those of rheumatism, are observed in some cases, though, as few ever prove fatal, little is known about the morbid anatomy.

Symptoms.—A period of about four days' *incubation* is followed by an *abrupt onset*, usually initiated by a chill, though in children convulsions may be the first indication of its presence. The *temperature* now rises rapidly, reaching, in some cases, as high as 107° or 108° F., the *pulse* running at from 120 to 140 beats per minute.

There is severe *frontal headache* (with photophobia, lachrymation, and flushing of countenance), *pain* in the back, limbs, and joints, with or without nausea and vomiting. After about twelve hours, the pains in the limbs, back, and joints become very much aggravated, lancinating pains shooting from the lumbar region down the course of the sciatic nerve, and along other large trunks. The lymphatic *glands* take on inflammatory action early in the course of the disease, the swelling and tenderness beginning in the inguinal glands, and soon afterward appearing in the axillæ and neck, these parts now being exceedingly sensitive and painful. The epididymes are also involved in a similar state, becoming swollen, sensitive, and painful. The muscles and soft tissues become tender to the touch, all the *joints* (both large and small) being reddened and swollen. The fever continues unabated for from one to five days, when it terminates in crisis. In many cases, a transitory, erythematous *rash* now appears, beginning on the palms of the hands and neck, and spreading over the entire body. This is liable to appear about the fifth or sixth day.

The decline of the fever, however, is deceptive. It is really only a *remission*, which lasts from two to five days, when a second paroxysm of *fever* occurs, attended by all the previous muscular and arthritic pains, headache, etc. But this paroxysm is less severe than the first, and a termination by crisis ensues in two or three days, and permanent *convalescence* now follows.

The disease is very prostrating in its tendencies, and convalescence is slow, from mental and physical debility. Colliquative sweats, diarrhœa, and epistaxis often occur during the remission.

Diagnosis.—Dengue may be confounded with remittent fever, as it usually occurs in malarious regions; but the persistency and severity of the muscular pains, and the glandular enlargement, with the cutaneous eruption, will be distinguishing features. The fever preceding the arthritic pains, and the erythematous rash, will distinguish it from inflammatory rheumatism, which it resembles; and,

if this be not sufficient, the glandular enlargements will be further distinguishing features. In its course, it resembles relapsing fever, but it differs from this in the fact that it is a disease of the interior, while relapsing fever is a disease of sea-ports, and lacks the marked swelling of the joints and lymphatics, as well as the eruption, which characterize dengue.

Prognosis.—Though an apparently alarmingly severe disease, the prognosis is almost always favorable, only those of extreme old age, or very young infants, succumbing.

Treatment.—The treatment will be directed toward a modification of the severe febrile disturbance and its accompanying unpleasantness. A remedy especially adapted to the picture presented by the symptoms is *jaborandi.* This should be given in small doses (two or three drachms to four ounces of water; dose, a teaspoonful every hour), repeated sufficiently often to moisten the skin, control the pulse, and lower the temperature, when the severe pain will be mitigated. *Phytolacca* may be added for the lymphatic inflammation, the jaborandi being combined, as, for instance, ℞ Specific jaborandi ʒiii, specific phytolacca ʒi, water ʒiv. M., order a teaspoonful every hour. When delirium is marked, *gelsemium* or *rhus tox.*, selected with regard to special indications, may be added to the treatment already prescribed, or employed separately.

Whenever practicable, a *vapor bath* or two, repetition being made available of a few hours after the preceding one, ought to do much toward relieving the force of the onset, and conducting the case through a mild course. The alcoholic vapor bath may do here, though the cabinet vapor bath is preferable, when at hand.

Attempts should be made to modify the severity of the pains with *macrotys, rhamnus californica,* or *phenacetin.* Opiates may seem to be demanded, but they should be avoided in all ordinary cases. In malarious districts, the judicious use of quinine may sometimes prove beneficial, especially if periodicity be marked.

Careful nursing should signalize the period of convalescence, until the patient has regained his wonted vigor.

XV. DIPHTHERIA.

Synonyms.—Angina Maligna; Angina Suffocata. German, Bräune Pruna (glowing coal). Spanish, Garrotillo.

Definition.—An acute, infectious disease, characterized by the exudation of a membrane upon a recently irritated surface—usually the tonsils and adjacent parts—the membrane containing the Klebs-Löffler bacillus, the disease being attended by blood-poisoning from ptomaïnes generated, resulting in profound prostration and anæmia,

with liability to extensive phagedena of the parts locally affected, or to paralysis of various organs and muscles, as well as pulmonary complication.

Historical Note.—Diphtheria is a disease which has been known from the days of antiquity. Asclepiades, who lived one hundred years before Christ, performed laryngotomy for the relief of obstructed respiration, and it is therefore probable that he treated membranous croup and diphtheria. Aretæus, a Greek physician, who lived at the beginning of the Christian era, described mild and severe cases of diphtheria clearly, and Galen, who lived in the following century, wrote vividly of a fatal disease characterized by the coughing, hawking, and spitting of a membrane. Cœlius Aurelianus, and Aëtius, the latter of whom lived in the fifth century, described it also, in unmistakable terms. No literature upon the subject exists to show that it prevailed during the Dark Ages, but this must be ascribed to the dearth of written records made during that time. It is evident that it occurred in severe epidemics in the sixteenth century, and from then to the present day an unbroken chain of testimony exists to show that it has remained as one of the most fatal scourges of human life. It has seemed to travel from the east westward, the disease probably having been brought to this country by Europeans. It it believed that the first cases occurred near Boston, about the middle of the sixteenth century (1638 to 1663). The modern name "diphtheria" was first applied by Pierre Bretonneau, of Tours, France.

Etiology.—The disease is endemic to most large cities, prevailing epidemically at certain periods. It is not confined to cities, however, it sometimes occurring in rural districts, with great virulence. It is contagious, the infection probably being communicated through the membrane, both moist and dried particles being infectious, the virus possessing a remarkable tenacity of life. Modern microscopical investigators have been inclined to the opinion formed by Klebs in 1883, and indorsed more recently by Löffler, that the active principle of diphtheria is a germ, found in the diphtheritic membrane, which is described by bacteriologists under the name "Klebs-Löffler bacillus."

This is a non-motile *bacillus*, about the third of the diameter of a red blood-corpuscle in length, and about two and a half times as long as broad. It is rounded at each end, and somewhat enlarged, having a dumb-bell appearance. It contains no spores that are visible. It stains with alkaline methylene blue, and thrives in blood-serum, bouillon, milk, and on raw potato. It is very tenacious of life, having been known to retain its vitality for five months, when

the membrane was wrapped in a dry cloth; and when stained, it resists the bleaching power of acids. The ptomaïne generated by this bacillus, as well as other septic processes arising from changes occurring under the diphtheritic membrane, probably give rise to the grave symptoms which often attend.

The most recent observations tend to throw much *doubt* upon the identity of the Klebs-Löffler bacillus as the specific causal germ of diphtheria. In numerous instances, a similar microbe has been found in various situations—in the buccal and nasal cavities, tonsillar crypts, etc.—in healthy individuals, apparently the same kind; thus affording strong evidence of its non-malignant character, and necessitating further inquiry before the question is fully settled.

Sewer-gas has been supposed to be an active factor in the causation of the disease, but knowledge of the fact that the virus possesses great tenacity of life, and that it has been common practice to empty cuspidors containing sputa of diphtheritic patients into the drains, will account for contamination arising from sewers, whenever traps are faulty. The fact that houses where the disease has been, remain points of infection for so long, is easily explained by the knowledge that the virus retains its vitality for a long time when dried, and that it may become a portion of the dust which may finally settle upon the walls, to be afterward distributed by a commotion in the atmosphere, and become implanted upon a receptive surface, such as the sensitive throat of a child. Lack of caution in the disposal of carpets and bedding which have been about a diphtheritic patient, may incline to the same result.

Bacillus Diphtheriæ.

Direct contamination, from one diphtheritic patient to another, is also frequent. In mild cases, children are often about among their companions with diphtheritic throats, and the contagium may carry death to a susceptible person when the one communicating it may not be very severely affected. Although it is a disease which is not very widely diffused by one affected (a few feet of distance affording safety), the indiscriminate use of drinking utensils among children of a community or school, and especially among those of a single family, affords ready means for the spread of the disease. The fresh membrane, when implanted upon the mucous membrane of an unaffected person, seems often to possess particular virulence; and physicans and nurses frequently lose their lives by the communication of the disease from children, while attempts are being made to treat an affected throat, the gagging, coughing, and "spluttering" of a fractious

patient serving to project a particle of membrane with sufficient violence to implant it upon some receptive mucous surface of the operator.

Animals are supposed, by good authority, to be a medium of communication of the disease. It is certain that mammals and fowls are often affected with rapidly fatal diseases, manifested principally by membranous exudation in the throat. I have seen many cases of the kind among chickens in California, and it is said to be a common disease among other domestic fowls. Calves and cats are subject to membranous throat diseases, though it is claimed in certain quarters that these are not communicable to the human family. But it has been observed, in several instances, that severe outbreaks of diphtheria have been preceded by such a disease among fowls, when no case of the trouble had been observed for a long time before. The rapid flight of pigeons—their wide circle of haunts—would therefore suggest a cause for the distribution of the disease to great distances in rural communities. Dr. M. W. Taylor (London) observed a case in 1888, in which a young man, from no other apparent possible cause, was taken violently ill with diphtheria four days after cleaning out a pigeon-loft; and he came to the final conclusion that the disease must have resulted from infection from sick pigeons. In 1884, upon the island of Skiathos, off the coast of Greece, diphtheria appeared, where it had not been known for a period of at least thirty years before, under the following circumstances: During that year a dozen turkeys were introduced to the island, two of them being sick when they were taken there. Seven out of the dozen soon died with the disease, which was evidently contagious; three recovered, and two were sick at the time of the inquiry. In two of the fowls, a pseudo-membrane was found upon the laryngeal mucous membrane, and in one that recovered there was paralysis of the feet. During the time of the sickness of these fowls, diphtheria arose among the inmates of the house adjoining the inclosure in which the turkeys were confined, the prevailing wind being favorable to waft the emanations in that direction, and an epidemic of the disease occurred, lasting five months, one hundred and twenty cases occurring in a population of four thousand, with thirty-six deaths. Many other instances are on record, conclusively proving that diphtheria may be communicated to the human family through the medium of fowls.

Diphtheria is essentially a disease of children, and though older persons may be affected, the gravity of the case usually depends upon the age of the patient; the younger the child, the greater the danger, other things being equal. It is asserted that new-born chil-

dren possess a certain immunity, but this is probably due to the fact that they are not so liable to come in contact with drinking-cups, spoons, and other utensils apt to be used in common by other members of the family, and are protected largely from probabilities of outside contamination. However, new-born children are frequently attacked, especially in hospitals, and usually with fatal result, the throat difficulty being attended or preceded by phlegmonous inflammation of the umbilicus.

While adults usually resist the disease in ordinary epidemics and recover, aggravated epidemics occur where it is fatal to nearly all with whom it may come in contact. Another circumstance where it is singularly fatal is that where the fresh virus is implanted upon a mucous surface, as often occurs to physicians while manipulating the throats of children for diagnostic or therapeutic purposes. A prominent physician of Oakland died within a few days, a few years ago, from an accidentof this kind occurring while performing tracheotomy upon a desperate case in a child. Such cases are not at all uncommon.

Pathology.—The disease manifests itself in a variety of ways. A more or less extensive destruction of tissue attends the location of the membrane, this usually being the fauces, though other parts, as the nares, eustachian tubes, middle ear, larynx, trachea, lungs, mouth, œsophagus, or stomach, may occasionally be the seat of the exudation. Occasionally the eyes may be the point of destructive inflammation, either from extension of the disease through the nasal duct, or as the result of direct contamination. In hospital practice, it has been found that diphtheria is liable to locate itself upon the raw surface left after the operation of circumcision, with resulting destructive inflammatory action.

The exudation varies much in extent, sometimes covering but a small surface, and at others involving large areas, covering the entire fauces, uvula, and pharynx, and extending throughout the nares, or perhaps into the larynx and trachea. Dr. J. Lewis Smith records a case (Keating's Cyclopœdia) in which a cast from a considerable section of the lower bowel was voided by an adult patient, under his observation. The thickness varies from the eighth of an inch to as much as a third of an inch, in some cases.

The cause of the most severe and dangerous symptoms is the systemic poisoning arising from the ptomaïne generated by the specific bacillus, though septic ferments also arise from pent-up necrotic fluids confined under the membrane, in the majority of cases, without doubt, which complicate and add to the constitutional gravity of the disease. In some cases extensive sloughing of tissue results

at the point of location of the membrane, but this would not be serious in character, were it not for the systemic effects of the poison. In fact, the local symptoms cannot be considered a criterion of the seriousness of the case, as some which exhibit but slight local disturbance may result fatally in a short time, from heart failure.

The diphtheritic poison in the membrane induces a necrosis of the cells with which it comes in contact, and a blackened line is found about the borders and under the surface of the exudation in a few days after its appearance. The superficial cells and leucocytes are first attacked, then the deeper structures, a coagulative process or "hyaline transformation" of the dead structures succeeding, which results in the formation of the leathery (sometimes pultaceous) membrane. Foci of necrosis proceed inward from the surface, and become localized in various internal organs, such as the bronchial and mesenteric glands.

When completed, the *membrane* is found to consist of a delicate interlacing network of fibrin, containing epithelial cells more or less altered, leucocytes, nuclei, mucus, and amorphous matter, as well as the Klebs-Löffler bacillus, streptococci, and staphylococci. In a few days after its formation, decomposition begins. During the active stage of the disease, the membrane reappears with remarkable rapidity upon forcible removal, a few hours sufficing to replace it entire, as firm and extensive as ever. When the membrane is removed, a raw, bleeding surface is left.

The *kidneys* and *lungs* are notably the seat of pathological disturbances in diphtheria. Albuminuria is a frequent complication, and though this may arise from feeble action of the heart, obstructed respiration, or fever, the *direct* action of the diphtheritic poison upon the structures of the kidneys, is most apt to be the cause of the renal complication. We then have parenchymatous inflammation of greater or less degree, followed by hemorrhagic infarcts, glomerulitis, disseminated inflammatory action, with cell infiltration and disintegration. The epithelial cells lining the tubuli uriniferi become broken down and separated, forming casts. In the lungs there are evidence of pulmonary apoplexy, disseminated extravasations, capillary bronchitis, and infiltration of the alveoli.

Capillary hemorrhage and fatty degeneration of the cells may occur in the *liver*. The *spleen* may be swollen, so as to distend its capsule to the utmost, the pulp protruding upon rupture or slicing of its covering. There is softening of the pulp, with extravasations of blood into its substance and hyaline degeneration of its vessels, in protracted and severe cases. Extravasation of blood occurs in the *heart*, under the pericardial and endocardial surfaces, with degenera-

tion of the muscle-nuclei. The *lymphatic glands* of the cervical and submaxillary regions are swollen, and contain evidence of histological change. There are hyperæmia of the cells, hemorrhagic points in the periglandular tissue, and distribution of necrobiotic foci in various places. Hyaline degeneration is also observable at various points in the glandular tissue. The *bronchial glands* present evidence of similar changes.

The *blood* is darker than normal, and there seems to be a deficiency in the amount of fibrin, coagulation being imperfect. There is a notable increase in the number of white corpuscles, with evidence of débris of broken-down red corpuscles, as seen under the microscope. Extravasation of blood occurs in the brain and its meninges, as well as in the lungs, spleen, and kidneys.

Nature.—The nature of diphtheria is peculiar in many respects. It may be primary or secondary, usually occurring as a primary disease, but infrequently appearing as a secondary affection in scarlatina, and occasionally in typhoid fever, small-pox, measles, and whooping-cough. When it appears in these diseases, a marked aggregation of symptoms is observable, and the membrane will be found upon the surface of some point of irritation—the fauces in scarlatina, probably the larynx in pertussis. The complication is a grave one, usually proving fatal.

When occurring in patients in whom there already exists a local inflammation, the membrane usually appears upon the irritated surface. In scarlatina, where the fauces are the seat of irritation, the membrane is found upon its appearance. In coryza preceding a diphtheritic attack, the membrane is likely to be located upon the Schneiderian membrane. If conjunctival irritation precede it, the eye is liable to be the point of location. Eye hospitals have been notoriously the place of resort of diphtheritic conjunctivitis. Circumcision of the prepuce among children in hospitals has been so frequently followed by the location of diphtheria in the part afterward, that it has been considered advisable to substitute the operation of stretching the prepuce instead of incising it, for the purpose of avoiding this danger. When a blister is applied to the surface of the body in a severe case of diphtheria, the abraded surface is soon covered with membrane exudate.

A point to which much discussion has been given is, Is diphtheria primarily local or constitutional? A considerable number maintain that the membrane forms first, and that the ptomaïnes are afterward absorbed, rendering the disease constitutional after the membrane has been located for a time. But it is observed that the membrane speedily returns upon its removal during the active stage

of the disease, and it would seem from this fact that it is an effect, rather than cause, of the constitutional state. The long incubative period usual to ordinary cases also suggests constitutional contamination prior to the appearance of the membrane. In some cases, the constitutional symptoms are marked before the membrane appears. The system succumbs rapidly in severe cases before the poison from the membrane could apparently have time to act, were the constitutional symptoms deferred to the time for absorption from the membrane to cause them. Albuminuria and nephritis are often present on the first day in severe attacks, and it would hardly seem that such remarkably rapid results could follow the first appearance of the membrane.

Symptoms.—The stage of *incubation* varies from two to twelve days. Where the disease is communicated by inoculation, it appears usually within two or three days after the introduction of the virus; where it originates in the ordinary manner, it varies from seven to twelve days. It is observable that when the stage of incubation is short the disease is severe, while in those cases in which this is protracted, it is mild.

The *constitutional symptoms* usually appear simultaneously with the advent of the membrane. Sometimes these are altogether absent, and the local symptoms are all that exist to indicate the presence of the disease. There are few cases which do not exhibit more or less constitutional symptoms early in the course of the affection. The ordinary febrile invasion often marks the beginning of the disease, such as chilliness, followed by fever of considerable height, the temperature rising to 105° or thereabout, during the invasion stage. Sometimes, in young children, the disease is ushered in with vomiting or convulsions. Where the febrile condition is ushered in with a chill, the temperature usually runs a higher course than when the disease comes on insidiously. In the former case, the temperature is liable to reach 105° F. numerous times during the course of the disease, while in the latter case, a temperature of 103° F. is seldom reached.

There is no correspondence between the local and constitutional symptoms. In some cases the fever may be very slight and the membrane spread quickly from the start, with rapidly fatal results, while in others the fever may run high, and the local manifestation be limited to a small patch of membrane upon one of the tonsils. The temperature, if high, is liable to fall, after the first two or three days of the disease, to near normal, and the membrane may spread rapidly, while there is little or no pyrexial excitement. Later, however, there is almost certain to be a rise in the temperature,

probably due to a systemic infection, different from that of the pure diphtheritic virus, doubtless from purulency developed about the location of the membrane, or from some local inflammatory action, such as nephritis, tonsillitis, or pharyngitis.

But however near normal the temperature may be in marked cases, the *pulse* indicates profound constitutional disturbance, either by irregularity, or pronounced acceleration with feebleness, or by both. In many cases the pulse will be feeble, small, and rapid throughout the disease, sometimes, in young children, running as high as 170 per minute. In other cases it may be rapid in the start, but fall 40 or 60, within a day or two. In still other cases, it may be intermittent or remittent throughout the course of the disease, suggesting grave results from the beginning.

The *tongue* hardly ever presents evidence of morbid condition of the stomach or circulating fluids, as in some other infectious diseases. It is usually moist and slightly furred, but commonly presents no marked indication for remedies as suggested by specific tongue indications in certain other cases.

The *urine* will often be markedly scanty early in the disease, and if it be tested for albumen, it will be found loaded with this substance; though renal complication is not universally present.

Local Symptoms.—Within twenty-four hours after the beginning of the disease, where it affects the fauces or neighboring parts, there will probably be found some enlargement, tenderness, and redness of the tonsils; and inspection of the fauces at this time will detect the diphtheritic *exudate*, beginning to form over the anterior surface of these organs, first appearing as a small patch of ashen-gray membrane probably, but spreading rapidly, and often extending to the uvula and posterior wall of the pharynx. At first it may be difficult to distinguish this from the exudate of follicular tonsillitis, as this may run together from two or more lacunæ in some cases, and form patches of considerable size. However, this may be removed without much difficulty, while that of diphtheria is firmly attached; after the membrane has spread upon the uvula, there can be no confusion in this direction, as the exudation of tonsillitis is not found except upon the tonsillar surface. The tonsils and fauces soon become congested and œdematous, and the cervical lymphatics enlarged and painful, the neck being swollen and stiffened. In a few days, the membrane becomes *necrotic*, exhaling an offensive odor and coming off in shreds of dark gray or black masses of decomposing material, this occurring, in many cases, at the end of a week. The time of the detaching of the membrane varies, however, and it may remain two weeks or more before falling off. The surface covered by it is raw and bleeding, after its removal.

The *odor* of diphtheria, when the case is a severe one and there are marked putrefactive tendencies, is characteristic, and offensive in the extreme, resembling that of the recent excrement of chickens most markedly, though exaggerated and more offensive still.

There are *two varieties* of diphtheria, as regards the exudation, one in which there is a tendency to rapid development of membrane without much putrefactive change, and another where there is a tendency from the start to necrotic changes in the membrane, and breaking down of tissue, accompanied by offensive odor of the exhalations, without so much disposition to rapid advance. In the first case, the danger seems to be principally that of asphyxia, from blocking of the respiratory passages, while in the other it lies more in the extreme exhaustion which soon results.

Nasal diphtheria is sometimes insidious at first, the membrane forming out of sight in the nasal cavities, all the general symptoms of the disease appearing without the local evidence of the cause of the trouble. It usually extends to the pharynx after a time, the membrane appearing on the posterior pharyngeal wall, or on the pillars of the fauces, or tonsils. It may extend along the eustachian tubes and give rise to inflammation and destruction of the middle ear, with perforation of the membrane. As the exudation spreads along the nasal mucous membrane, the nostrils become obstructed, the patient breathing through the mouth, and speaking in a "throaty," muffled tone. A sanious coryza soon develops, which is excoriating to the lip, and, as the disease progresses, epistaxis frequently occurs. Where the membrane invades the eustachian tubes, there is tinnitus aurium and sticking pains in the ear, aggravated by swallowing, with more or less permanent loss of hearing.

When the *œsophagus* is invaded, there is dysphagia, with regurgitation of fluids and frequent vomiting, the vomited matter containing, as the disease progresses, portions of membrane. Later, portions of the membrane may appear in the stools.

Sometimes the vagina, rectum, or labia are invaded, and there may be pain, tenderness, swelling, and redness over the inguinal glands.

Pharyngeal diphtheria may extend to the larynx and trachea, or *laryngeal* diphtheria may be developed independently, when the membrane begins in the nares. It is most apt to occur in young children; the younger the child, the more liability to this form of the disease. A croupy cough soon becomes prominent, respiration being rough, and the voice raspy and indistinct, soon falling to a whisper. The dyspnœa is marked, the auxiliary muscles of respiration being taxed, paroxysmal attacks of difficult breathing occurring

at frequent intervals. The supra- and infra-scapular spaces sink during inspiration, cyanosis becomes marked, and stupor or extreme restlessness becomes pronounced, as dissolution approaches. Death by suffocation finally results. Pulmonary changes are evidenced by areas of dullness and absence of the respiratory murmur, with submucous, subcrepitant, crepitant, and sibilant râles intermingled. The epiglottis, vocal cords, and interior of the larynx become completely covered with exudation, which, in many cases, extends far down the trachea.

Paralysis.—This is a distinctive feature of diphtheria, appearing to greater or less extent in almost every severe case, doubtless being the cause of death in many instances of heart failure. Careful investigation of the nervous system has been made, to determine what the pathological lesions, if any, were, and it has been claimed by some authors that degenerative changes in the nervous structures account for the condition. Charcot and Vulpian, in 1862, detected granular degeneration of the nerves of the soft palate. Oertel, in 1871, found extravasations in the substance of the brain, spinal cord, and spinal nerves; in one case where death had occurred from diphtheritic paralysis. Buhl found a similar condition, and, in addition, determined that the nerves were thickened at their roots, and that their sheaths were filled with lymphoid cells and nuclei.

However, this does not prove that diphtheritic paralysis is occasioned by such changes in those who recover, and from whom the paralysis spontaneously disappears in a few months. The fact of speedy spontaneous recovery militates against the proposition that structural changes necessarily operate in producing them. It seems apparent that the paralysis may be the result of the depressing action of the diphtheria ptomaïne on the functional activity of the nerve centers. The most common point of paralytic exhibition, except the heart, perhaps, is the group of muscles about the fauces and pharynx. Difficulty of articulation and swallowing are here the prominent symptoms, the uvula hanging down and the epiglottis losing its reflex action, attempts at deglutition being attended by the regurgitation of fluids through the nose, solids causing much struggling and difficulty, when attempts are made at swallowing. Paralysis of the laryngeal muscles, with aphonia, usually attends this condition. These symptoms come on late in the course of the disease, when the membrane is disappearing, or a week or ten days afterward. The paralysis of the epiglottis may endanger the lungs, through liability of portions of food to pass into the larynx, and, as sensation as well as motion is gone, reflex coughing is not thus excited. Expectoration is impeded, and the pharynx may be blocked

with tenacious mucus, which the patient lacks the power to remove. As the pharyngeal paralysis disappears, other muscles of the body may become involved, the lower extremities being most liable to suffer. There is no regularity of the symptoms, however, a hand or arm, a foot or leg, the muscles of the neck, the orbicular muscles or the lower sphincters, all being liable to paralysis. Cardiac paralysis is also liable to occur during convalescence, and sudden collapse may take place upon too sudden exertion, many days after the patient seems out of danger.

However, paralysis of this kind usually subsides spontaneously, time and care being about all that are necessary for complete recovery of sensation and motion in the affected part.

The *bowels* seem to escape serious disturbance in this disease, unless, as happens in rare cases, the membrane locates upon some portion of the intestinal mucous membrane. Usually there is no disturbance in their functions—a favorable condition for recuperation after the debilitating disease has spent its force, surely.

Diagnosis.—The diagnosis of diphtheria is not always a simple matter. Pseudo-membranous exudations often occur upon the laryngeal mucous membrane which are not diphtheritic; for though they may cause death in a short time by asphyxiating the patient, they do not infect the system with the profound constitutional poisoning of that disease, nor do they present other clinical symptoms of diphtheria, such as the peculiar stench, contagiousness, and paralytic sequelæ, when recovery results. Also, bacteriologists are somewhat confused as regards the evidence afforded by the Klebs-Löffler bacillus, as it is asserted that there are cases of angina attended by an exudation which contains a bacillus identical, morphologically, and in its behavior on culture, with this germ, which does not communicate diphtheria when inoculated.

Clinical characteristics are the best criterion by which to determine the identity of the disease, in general practice. The extreme prostration, the markedly feeble and rapid pulse from the start, the putrid odor, in conjunction with the characteristic leathery membrane, can hardly be mistaken for any other disease, even if it should be attended by angina with a pseudo-membrane. In large cities, where diphtheria has become established (and this is the case with almost any city possessing a sewerage system), the chances are all in favor of any angina attended by the formation of a leathery membrane in the throat being diphtheria. The exceptions are certainly rare, and are only worthy of notice to complete the requirements of a text-book on practice. Pseudo-membranous croup lacks the prostration that marks true diphtheria, is not attended by the rapid, fee-

ble pulse, and is free from the stench that characterizes disintegration of the diphtheritic membrane.

Prognosis.—The prognosis of diphtheria is always doubtful when the poison is sufficiently intense to markedly disturb the vital functions. Even though the membrane may not be extensive, the constitutional effects may result seriously, as there are so many avenues open to a fatal result. If the prostration be not marked at first, there is always a possibility that the kidneys may become involved so seriously as to destroy the patient. Then, if this danger be past, there is still danger that heart failure may suddenly terminate the case. It being a treacherous and uncertain disease, care must be observed not to pronounce too favorably, in any event.

It is to be remembered, however, that there is great difference in the character of different epidemics, and that a prognosis may depend considerably upon the epidemic influence at hand. Mild epidemics are attended by small mortality, and the treatment that seems to make little impression in severe cases may suffice at such times.

The *age* of the patient, also, will exert an important bearing upon the prognosis, the younger the subject the greater the danger of laryngeal complication, probably one of the most serious conditions liable to attend.

The *period* of the disease determines to a certain extent the character of the danger. During the first six or seven days laryngeal complication, or septicæmia, is most liable to appear. The voice should now be watched, to determine whether it becomes husky or croupy. A throaty voice, with snuffling breathing, will suggest nasal complication, another portentous sign, as such cases seldom recover. Rapid prostration, with lividity of countenance, delirium, or tendency to drowsiness, especially if the pulse be irregular and the temperature elevated, will suggest septicæmia. After the first six or seven days, inflammatory complications, if these exist (such as nephritis or tonsillitis), have become fully developed, and the danger may now be in this direction. Or, at this time, sudden death by syncope is liable to result from heart-clot, or abrupt arrest of cardiac action from other causes. After the second week, nervous symptoms, especially paralysis, may be expected, though if the patient has reached this period, the prognosis may be considered more favorable, as the paralytic symptoms are not liable to prove fatal unless the heart becomes involved.

Treatment.—The patient should be *isolated*, and a strict quarantine established between the sick-room and all outsiders, except the nurses and physician. Carpets and superfluous *fomites*, such as

rugs, lace curtains, and extra bedding, should be removed. Discharges from the mouth, throat, or other parts liable to contain diphtheritic matter, should be disinfected with a strong solution of corrosive sublimate before being emptied, and then should be buried in a trench dug for the purpose; or, when possible, it should be received from the patient on cloths, these to be immediately burned. A basin of weak carbolic-acid solution should be near the bed for washing the sponges, etc., used about the patient, and, instead of pocket handkerciefs, cloths, which may be immediately burned, should be employed. Care should be observed to keep all feeding-cups, glasses, and spoons separate, and these should be cleansed in an antiseptic solution before being allowed to leave the room, to be washed. Linen, over a piece of waterproof made into a bib, to pin over the nightdress of the patient, will serve as a protection against the irritation of the neck and throat from the acrid discharges which are liable to excoriate the skin, if coming in contact with it.

At the termination of the case the room and its contents should be thoroughly fumigated by burning sulphur, the air in the room being moist at the time, or else the walls and floor should be diligently scoured with a strong solution of corrosive sublimate, and the bedding and other clothing well boiled in a carbolic solution.

When possible, experienced nurses should be in charge of every case of diphtheria, as intelligent precautions against the spread of the disease are as important as attention to the patient in hand; and the life of the patient may depend largely upon proper care at such times, especially after the operation of tracheotomy, prompt feeding and other details now being especially important.

Physician and nurse should be careful about keeping the mouth closed while standing over the patient, and in treating the throat or making examinations it is wise to wear a mask or silk handkerchief over the mouth and nose, as a safeguard against accidental infection. The hands of the attendant should be cleansed frequently, in disinfectants, those of the physician especially before leaving the room after examining the patient, and those of the nurse especially before taking meals.

It is an excellent plan to see that the room is constantly permeated with steam from an antiseptic and aromatic solution. The following prescription is highly recommended by Dr. J. Lewis Smith, and I have used it with considerable satisfaction: ℞ Carbolic acid and oil of eucalyptus, aa, ʒi; spirits of turpentine, ʒviii. This should be mixed, and a tablespoonful of it mixed with a quart of water for use, in a shallow vessel, which is kept constantly simmering upon a gas or kerosene stove. This tends to soften the exudation and

encourage secretion of the mucous membranes, thus assisting in throwing it off.

Ventilation must not be neglected, as it is especially important here, on account of rapid vitiation of the air from putrescent odors and emanations.

It should be remembered that syncope and sudden death are not uncommon in this disease, and that the upright position is inclined to promote such an accident. The *recumbent posture* is the safe one for the patient until convalescence is well established. I have known of several cases where dangerous syncope resulted from incautious getting up before the disease was fully under control, and even during early convalescence.

The *medicinal treatment* of diphtheria is still very unsatisfactory in its results. It is true that many mild cases seem to do well on *aconite* and *phytolacca*, but these would probably recover if left alone therapeutically, and carefully nursed. Doubtless phytolacca relieves the congestion about the fauces to a certain extent, and is therefore of some use—this being its specific province; and it will be called for where there is considerable tonsillitis. But when we encounter severe cases, we are frittering valuable time away, when we depend upon it, in the least, as a remedy for diphtheria, as its effects can be but illusory. The "special sedatives" are also subject to objections, as they can exert little or no control over the course of the disease, and though pyrexial action be present, it is not so detrimental as the after-effects of any remedy which can act as a cardiac depressant in minute doses, where there is so much inevitable prostration. Aconite, veratrum, gelsemium, and jaborandi should be tabooed here, as there is no rational room for their exhibition. If a sedative remedy is to be employed especially for its sedative effects, there can be no objection to *ferric phos.* 3x, which is very reliable for the general purposes of a sedative, and cannot produce bad results. The markedly anæmic condition would rather favor the theory of its application.

We seldom find the antiseptic propositions, manifested by the *tongue*, which are found in some other acute infectious diseases. The gastro-intestinal canal does not seem to become sufficiently disturbed to develop marked tongue symptoms, and when the disease is ushered in by vomiting, it is usually caused by an effect upon the nervous centers produced by the diphtheritic poison, rather than by morbid accumulations or local irritability. I have never yet seen a case where the sulphate of sodium, rhus tox., sulphurous acid, hydrochloric acid, or any other specifically tongue-indicated antiseptic was pronouncedly called for. Where this was the case, I

should administer the proper one with faith that some good might be accomplished. Chlorate of potassium has seen its day, as the common complication of nephritis is well recognized, and also the fact that this drug is very liable to produce a similar condition without other causes. It is so detrimental as to be considered dangerous, by modern therapeutists. The disease is *necrotic* in its tendencies, and should not be furthered in its effects by strong chemicals which are liable to favor destruction of red blood-corpuscles and fibrin, as well as firmer structures.

Probably we at present possess but two or three ideal remedies for diphtheria; and, unfortunately, they are not always successful. However, *echinacea* combines nearly all the properties desirable for the fulfilling of the most important indications in the treatment of this disease, as an internal agent. It is a sedative, while it stimulates the vital forces at the same time. It is eminently antiseptic and anti-necrotic. Furthermore, it is undisputably harmless in its effects. There can be no danger in saturating the system with it, and this should be done throughout the disease, unless there be some prominent call for another remedy, the action of which might be embarrassed by it. In all cases where septic and necrotic tendencies are prominently marked, ten or fifteen drops of specific medicine, or green-plant tincture, every hour, to a child eight years of age, will be demanded. Where it is desirable to obtain the effect of some other remedy, this should be employed as an intercurrent, throughout the treatment.

Another remedy, which is better adapted to many cases, on account of its superior action in cardiac failure, is *lachesis*, an agent which has made many cures of severe diphtheria. My attention was called to it many years ago, under the following circumstances: A severe epidemic broke out in a neighboring township, while I was practicing in the country, and proved to be remarkably malignant in character, being fatal in almost every case affected; and it did not cease until entire households were eradicated, adults and children in common, though it was confined to a small neighborhood. Several families employed old-school physicians, but at last the disease entered a family of homeopathic proclivities, and a young homeopath, who had recently located in the neighborhood, was called. There were several members in this family, but all recovered except one, which was considered a remarkable circumstance, as the disease completely swept away several families which had been treated by allopaths. In conversation with the homeopath afterward, I learned that he had depended almost entirely on lachesis, in the treatment of his cases.

The remedy is so well adapted to this disease that I will reprint, from Hughes' Manual of Pharmacodynamics, an extract bearing on its action, both in this disease and others attended by local gangrene and systemic infection:

"Malignant local inflammation, with secondary blood infection and nervous prostration, have proved preëminently the sphere of lachesis. A typical instance is traumatic gangrene. Of this disease Dr. D. M. Dake has published three cases, which are so decisive as to overcome even Dr. Hempel's skepticism as to the virtue of the remedy. They are given at length in the second edition of his Materia Medica; and in the fourth volume of the *American Homœopathic Review* Dr. Searle, of Brooklyn, has recorded two others. To these I would add the testimony of Dr. Franklin, who, as army surgeon in the late civil war in America, had abundant opportunity of seeing the disease. 'I have used this remedy,' he writes in his Science and Art of Surgery, 'in a number of cases of gangrene following wounds, and have never been disappointed in its results. In a case of comminuted leg fracture, terminating in gangrene and threatening speedy destruction of the limb, the gangrene was quickly checked by the internal and external use of lachesis, the inflammation subsiding, and the healing process moving on to a complete cure. In another case of compound dislocation of the ankle-joint, with fracture of malleolus externus, followed by gangrene, lachesis effected a speedy cure, the patient making a good recovery under the surgical treatment employed. I cannot recommend too highly the use of this agent for gangrene, and am confident that the observations of all who have employed or may employ it will bear me out in the assertion that it is eminently curative of gangrenous affections.'

"It is affections of this kind, moreover, which form the bulk of the paper of Dr. Carroll Dunham, to which I have referred. He begins with a case of septicæmia occurring in his own person, as the result of a wound incurred during the post-mortem examination of a case of puerperal peritonitis. Both the local and general symptoms were severe, but they rapidly yielded to lachesis 12, three times a day. Next he relates an epidemic of malignant pustule, in which he treated eight cases with lachesis alone. 'It relieved the pain within a few hours after the first dose was given, and the patients all recovered very speedily.' Then he speaks of three cases of phlebitis supervening upon ulcers (probably syphilitic) of the lower extremities. There was great and sudden prostration of strength, low muttering delirium, and general typhoid symptoms, indicating pyæmic infection. The effect of lachesis was all that could be desired, the patient rallying promptly, and all symptoms of phlebitis speedily

disappearing. Last, he narrates one case, and refers to others, of carbuncle, in which the constitutional symptoms denoted very great prostration, *not* preceded or attended by the nervous and vascular erethism which is sometimes observed in similar cases. The absence of this condition, he thinks, in all these disorders, the indication for lachesis as against arsenicum, when the asthenia is not so complete as to call for carbo vegetabilis.

"Dr. Dunham finally refers to the usefulness of lachesis in certain cases of diphtheria. In these the tumefaction of the throat was slight, and the redness of the mucous membrane hardly noticeable, the diphtheritic deposits consisting merely of two or three patches hardly larger than a pin's head. But the prostration of strength was quite alarming; the pulse became, in a very short time, slow, feeble, and compressed; a cold, clammy sweat frequently covered the forehead and extremities; the breath was fœtid; the appetite entirely destroyed. 'In such cases,' he writes, 'in all in which the constitutional symptoms thus predominated over the local, lachesis produced prompt and lasting improvement, so much so that very rarely was any other remedy given subsequently.' To the same effect is the testimony of Dr. Tietze, of Philadelphia, in the fourth volume of the *United States Medical and Surgical Journal.* He mentions a purple, livid color of the affected parts, with dull, dry appearance and little swelling, also pain out of all proportion to the amount of inflammation, as local characteristics of the remedy. He places it third to belladonna and apis in throat affections, in the descent from sthenic to asthenic conditions. Dr. E. M. Hale also contributes to the *American Journal of Homœopathic Materia Medica* three similar cases of diphtheria in children, which made a rapid recovery under lachesis, while the rest of the family (altogether eight in number) under old-school treatment succumbed to the disease."

In using this remedy, I prescribe two or three grains of the 6x trituration, to be repeated every two hours.

But there is a form of diphtheria in which the tendency to early putrefaction and necrosis is not so marked as that of rapid spread of the membrane. We here have the danger of blocking of the larynx with exudate to encounter, especially in young children; and neither echinacea nor lachesis seems to possess the property of controlling plastic exudation. *Potassium chlor.* 3x comes nearer fulfilling this requirement than any other remedy we know of, and Schuessler has been very enthusiastic over its action as a specific for diphtheria, on this account. It certainly is of considerable service in this particular class of cases, as I know from experience, and here

we will administer echinacea or lachesis every three or four hours, and give potassium chlor., adding ten grains of the 3x to half a glass of water; dose, a teaspoonful every hour.

There are many other remedies which have been advocated for the internal treatment of diphtheria, such as *sulpho-carbolate of sodium, benzoate of sodium, pilocarpine, turpentine, euchlorine, tincture chloride of iron, etc.* The numerous remedies recommended by different authors are suggestive of the fact that few of them possess the required virtues, when desperate cases are encountered. However, a careful study of these is recommended, lest something useful be neglected.

The use of alcoholic *stimulants* throughout the disease is an old practice, but some adhere to it at the present day. Mild cases will recover under such treatment, doubtless, as they will recover spontaneously; but as alcohol does not cure anything else, it is difficult to believe that it will cure severe cases of diphtheria. Happily, it is not so fashionable a remedy as formerly, and one can now omit it from his treatment without losing caste among his allopathic neighbors.

The *local treatment* of the membrane is an important matter, at least is so considered, as this is liable to be the nidus of septic accumulation, and the source of septicæmic infection, quite independently of the original diphtheritic virus. To abridge the extent of this formation, lessen its thickness, and render it as little septic as possible, seem, then, important considerations. These are to be accomplished by the use of *solvent antiseptics*, such as *lime-water, pepsin, trypsin, papyotin*, and *peroxide of hydrogen*, as well as many others not here mentioned. The *douche* may be used to irrigate the nasal cavities in nasal diphtheria, though too much of this is liable to bring on inflammation of the middle ear.

Swabbing of the throat has fallen into disrepute in many quarters, as the struggles of the patient in resisting the operation, for which many children entertain a great horror, are liable to result in blind and forcible efforts, which irritate the tender and partially disorganized structures, inviting the deposition of more membrane, and aggravate what inflammation may already be present. *Atomization* of fluids with a spray apparatus is the best method of application, the remedies being employed in solution.

The following combination affords good satisfaction, the throat being sprayed with it frequently: ℞ Glycerine gtt. xx, carbolic acid gtt. xv, aqua ʒi, essence of peppermint ʒiss. Misce. Or, ℞ Oil of eucalyptus ʒii, benzoate of sodium ʒi, bichromate of sodium ʒii, glycerine ʒii, lime-water Oi. Misce. These may be used both for nasal and pharyngeal exudation.

Where the laryngeal exudation becomes so abundant as to impede the respiration, and is evidently advancing, tracheotomy or intubation should be resorted to early, before the patient has become so exhausted as to render the effort useless.

Inflammatory conditions must be met by proper special remedies. Tonsillitis may demand phytolacca; inflammation of the middle ear, piper methysticum or pulsatilla; nephritis, rhus aromatica or vesicaria communis.

The *paralysis* may be benefited somewhat by the proper application of galvanism and the internal use of nerve stimulants, but time usually relieves such conditions nearly as rapidly as treatment, and if patient and friends are assured that a favorable outcome may be expected, this symptom will not cause much trouble.

The food, during the disease and during convalescence, should be of the most nutritious character, and, at the same time, of a kind to be easily swallowed and digested. Meat juice, malted milk, beef peptonoids, and plain, fresh milk may all be resorted to, as is most convenient and acceptable. Food should be given in small quantity, often, as it is to be remembered we are dealing with a rapidly prostrating affection. Cathartics should be avoided, as they embarrass digestion, and derange the functions of the alimentary canal.

Time has favored belief in the antitoxine treatment, in *desperate* cases. There is no doubt that the injection of the substance into the circulation is fraught with considerable danger, for numerous cases are on record where it has been employed for prophylaxis, in which sudden death was the result. But, again, it has been followed by favorable changes, where death seemed imminent, and it seems indisputable that in its proper place it is sometimes surprisingly efficacious. The proper plan, it seems to me, is to hold it in reserve for cases which defy other therapeutic measures, and, when these arise, to employ it in combination with them. From correspondence with several of our best physicians, as well as from reading our current medical literature, I am convinced that we cannot afford to ignore it as a means of salvation, in occasional cases.

Several reliable brands of horse-serum are in the market, each package being accompanied by full directions. Where death has seemed imminent in a few hours, numerous cases of malignant diphtheria have convalesced within twenty-four hours after the hypodermic use of this agent; the temperature speedily falls, the membrane rapidly disappears, the symptoms of prostration pass away, and, though an erythematous rash, with cutaneous irritation, may attend, convalescence soon follows. However, it should only be employed in those cases which offer little other hope.

XVI. ERYSIPELAS.

Synonyms.—St. Anthony's Fire; Rose.

Definition.—An acute, contagious disease, excited by the streptococcus erysipelatis, characterized by a peculiar inflammation of the skin and subcutaneous tissue, attended by an irregular fever and tendency to rapid spread, with speedy resolution and liability to relapse.

Etiology.—The cause of this is undoubtedly local infection from a specific germ, which gains entrance to the tissues through some abrasion of the cutaneous or mucous surface. The disease was formerly divided into *traumatic* and *idiopathic* erysipelas, from the fact that it develops occasionally in wounds, and *seems* to sometimes develop upon a cutaneous surface where no abrasion of the skin has been made; but closer inspection will always show that there has been an opening in the integument, through which the germ has entered. The division, therefore, is manifestly illogical. The abrasion may amount to only a slight excoriation, such as a mosquito bite, a small pustule, an intertrigo, or some minute point that escapes notice until the erysipelatous manifestation is developed. A common place for the appearance of the disease is the face, and the first point of localization may be out of sight, upon the mucous membrane of the nose, mouth, eyelid, or ear, it spreading from there upon the skin, through one of the natural orifices. Or, it may arise at the genitals or anus, intertrigo, eczema, chafing, a pustule, an ulcer, or some other break in the skin, admitting the infection. Erysipelas may arise in the pharynx and traverse the eustachian tube to the middle ear, pass through the tympanum and appear on the face; or it may arise in the nose, and passing through the nasal duct, appear in the eye, to spread from there to the face; or it may pass from the nostril to the face, or from the pharynx through the mouth, and the disease seem idiopathic, when some abrasion, not observable, has allowed the streptococci to enter the tissues. Erysipelas is peculiarly severe and fatal in new-born children, though after six months of age it is not more severe, probably, than in adults. Puerperal women are also more than ordinarily susceptible to the infection. Vaccination provides a ready point for the entrance of the infection, and erysipelas is not an uncommon sequela of that operation. That the disease can be communicated from one to another, has been proven by the vaccination of several persons from one who shortly afterward developed it, all the others soon developing the disease also. This is one of the principal objections to the employment of humanized virus in vaccination. Some persons seem predisposed to yearly attacks of erysipelas, the disease returning about the same

period, for years in succession. The face is usually the point of attack here, rhinitis, eczema, acne, or some other abrasion of the skin affording it entrance. Erysipelas usually occurs sporadically, though epidemics occasionally develop.

(A) ERYSIPELAS COCCI. (IN THE SPLEEN.
(B) ERYSIPELAS COCCI. (IN THE CUTIS.)

It has been a disputed question whether the germ of erysipelas is a separate and distinct organism, similar to but not identical with the pus-streptococcus. Koch and others, after careful investigation, have decided that it is identical with the *streptococcus pyogenes*, while others, probably fully as reliable, declare that though the similarity is great, there is a distinction. From a clinical standpoint, the latter view seems the correct one.

Pathology.—An early infiltration of the skin and subcutaneous connective tissue is the first marked pathological alteration, though careful inspection will now find the streptococci occupying the lymphatics, where they are at first confined, whence they soon afterward invade the adjacent connective tissue. The skin becomes œdematous and sharply raised over the affected area, the part being at first bright-red, tense, and shining, though afterward becoming livid or brown, the epidermis now being thrown off in scales or flakes. The infiltration is serous or fibrinous in character, and contains an abundant supply of cells (leucocytes), which surround the vessels. The streptococci, which first appear in the lymphatics, soon invade the connective tissue of the skin, and, sparingly, the subcutaneous tissues, in chaplets or coherent masses, which constitute colonies, and around which necrotic changes, more or less marked, occur. The amount of exudation and necrotic change determines the severity and characteristics of the case. Where the exudation is moderate in amount, there is not very extensive destruction of tissue, scaling and flaking of the epidermis, with a more or less permanently established debility of the skin being left behind, manifested by a deepening of the color of the affected part, which may persist for some time after convalescence. Where the exudation is more abundant, blebs and vesicles rise on the surface, due to necrotic changes in the cells of the rete Malpighii, at numerous adjacent points, with subsequent deliquescence of the partitions, and liquefaction of the contents. This constitutes *erysipelas vesiculosum* or *bullosum*. Sometimes pus accumulates instead, constituting *erysipelas pustulosum*.

These dry up into scabs, becoming *erysipelas crustosum*, and if necrosis occur about them, we have *erysipelas gangrenosum*, the necrosis varying in extent markedly, in various cases. The mucous membrane of the respiratory tract may partake of the general characteristics of the cutaneous affection, the tissues of the lungs becoming infiltrated and occupied by streptococci, with the resultant changes. The pathological condition will differ from croupous pneumonia, from the fact that there is no plastic exudation into the alveoli, as in that disease. In severe and prolonged cases, the tissues generally undergo the general changes common to prolonged pyrexia. There is parenchymatous degeneration of the muscles, intestines, liver, spleen, kidneys, etc., though these changes are not pathognomonic of the erysipelatous disease.

Symptoms.—From three to seven days' *incubation* occur after the entrance of the streptococci, before the disease becomes fully developed.

Like some other infectious diseases, the actual attack is preceded by more or less marked prodromal symptoms, such as drowsiness, irritability, malaise, muscular pains, etc. The disease proper often begins with a *chill*, more or less marked, though in young children, convulsions or vomiting may replace it. A rise of temperature follows, the thermometer soon marking as high as 105° F. While irregular, the *temperature* is liable to remit slightly in the morning, with an evening exacerbation, though this rule may be reversed. It may not rise above 103° in mild cases, and it may reach 106° and higher, in severe ones, the temperature depending much on the extent of the local disease. As long as the local inflammation continues to advance, the fever continues high, though the advance usually ceases by the third or fourth day. About the fifth or sixth day it may fall rapidly to normal, though this cannot be depended upon. With rise of temperature, the pulse becomes correspondingly increased in rapidity, ranging, in adults, from 100 to 120 per minute.

The *local manifestation* is almost always a coincidence; an elevated, reddened point of localization is observed, which spreads rapidly, showing an abrupt elevation at its borders, and in which there are sensations of tension, burning, itching, tingling, and darting pains. The redness disappears upon pressure, leaving a pit in the œdematous tissue, but returns rapidly upon its removal, the part being sensitive to touch. Where the subcutaneous tissue is abundant, as about the eyes, the swelling is a remarkable feature of the disease, the eyes soon being swollen shut, their presence almost obliterated, and the countenance disfigured. During the active progress of the disease, while the fever remains high, it is accompanied, in many

cases, by severe constitutional symptoms, such as loss of appetite, nausea, vomiting, intense headache, thirst, and even delirium. The tongue becomes dry and brown, and is usually covered with a thick coating, which may be pasty-white. The *urine* is scanty, high colored, and often albuminous, and the *bowels* are constipated. Where the *face* and *scalp* are extensively affected, the patient may be delirious or comatose. Where the *mucous membranes* join the skin near the point of attack, they are frequently involved. When recovery terminates the case, the swelling gradually subsides, the redness disappears, the temperature declines, secretion becomes reëstablished, and the appetite returns. Where death ensues, the patient usually dies with a high temperature.

The disease manifests a marked predilection for the *face* and *scalp*, it being estimated that nearly seventy per cent of the cases encountered are located here. Where the scalp is deeply involved, a permanent, or at least a long-continued, alopœcia may result over the most severely affected surface.

In the *new-born*, the disease usually commences about the navel or the genitals. Imperfect healing of the navel may leave an opening for the entrance of the miroörganisms, and chafing about the privates, so common in very young children, is attended by excoriations, offering abrasions favorable for the ingress of the disease. The disease arises insidiously in these cases usually, only a slight blush indicating its presence, for three or four days. Finally, a high fever develops, and the local manifestation becomes observable. The skin is soon enormously distended and glistening, subcutaneous abscesses develop in many cases, and gangrene soon follows. The inflammation may extend along the umbilical vein, and peritonitis, with purulent infiltration, result. The child is extremely restless, cries constantly, refuses nourishment, finally becomes comatose, and dies in this condition, or in convulsions.

The disease is an acute one, and runs its course in from ten to fourteen days, usually, though it may leave serious sequelæ, which may persist for a long time. Among these are abscesses of the skin, gangrene, bronchitis, and pneumonia. Where the throat is severely involved, œdema of the glottis may arise. Cardiac affections, such as endocarditis and pericarditis, may ensue. Inflammation of the meninges sometimes results, and death may be caused by this affection, the disease extending through some of the foramina, where the head and face are involved. Eye affections, such as keratitis, panophthalmitis, and amaurosis, are of occasional occurrence.

Diagnosis.—The diagnosis of erysipelas is not difficult. The intense redness and swelling, localized, and usually known to arise

from a wound or break in the integument, the intense swelling, with abrupt border of the tumefaction, this being accompanied with high fever and other marked constitutional symptoms, can hardly be mistaken for any other disease. Rhus poisoning might present some of the local symptoms of erysipelas, but the severe constitutional symptoms would be absent. Other cutaneous affections would be subject to the same exception. Where there was any question in the clinical aspect, a microscopical examination might detect the characteristic streptococcus.

Prognosis.—The character of the surroundings will suggest much, as to the probable outcome of a case. In traumatic erysipelas, occurring in crowded hospital wards, the condition is always a serious one. Where the case is sporadic, and sanitary conditions are favorable, with a patient of constitutional vigor, the prognosis is not unfavorable. When the disease occurs in new-born children or puerperal women, a guarded prognosis is safest for the reputation of the attending physician.

Treatment.—A better knowledge of the etiology and pathology of erysipelas has not advanced the treatment of the disease, to any great extent. The best treatment we possess is an empirical one, though the general principles of dynamical therapeutics apply here, as elsewhere. The treatment may be divided into *constitutional* and *local*, the aim being to neutralize the ptomaïnes generated and the inflammatory action as much as possible, it not being probable that treatment directed to the destruction of the streptococci will amount to anything more than an aggravation.

It is well in the beginning of treatment to inspect the tongue carefully, to see if there be any prominent indication of blood-sepsis. One of two remedies will be indicated here, provided there is any call for remedies of this character. We may have the *sulphite of sodium* indication, suggested by the broad, flabby tongue, with pallid mucous membrane, covered with the pasty-white coating; and we may have the *sulphurous acid* tongue, indicated by the dark-red mucous membrane with brown coating, this being usually dry. Sometimes there is no prominent tongue indication, and the treatment is much simplified. For the sodium tongue, we will administer capsules containing sodium sulphite, 1 gr., every three hours. For the sulphurous acid tongue, twenty drops of this drug, well diluted, at about the same intervals.

Having seen to it that provision is made for sepsis as indicated by the tongue, we will devote ourselves to other treatment, nearly if not quite as important. The nature of erysipelas is to destroy connective tissue by necrosis. If we can mitigate this tendency, we shall

be able to modify the extent of the destructive action, lessen the spread of the disease, and protect the tissues from very severe inflammatory and degenerative changes, as well as ameliorate, considerably, the severity of the general symptoms. Our best remedy for necrosis of soft tissues is *echinacea;* and it should constitute a portion of the treatment of every case, being administered steadily throughout the entire course of the disease. It is not incompatible with any other remedy we may need, and can be administered in combination (in alternation, or in conjunction) with any other treatment. Ten or fifteen drops may be advantageously administered, to an adult, every hour during the height of the disease, and four or five times a day during convalescence, to guard against relapse.

A high temperature would suggest the combination of *jaborandi* with echinacea, three or four drops of the specific medicine every hour tending to reduce the temperature as well as the local inflammation, through its sedative action. Any one who has experienced the gratefully cooling influence upon the skin in his own person during fever or inflammatory action, can appreciate the benefit liable to be derived from this remedy in such a condition as erysipelas.

Rhus tox. is especially useful as a sedative where the tissues of the face are involved, as it seems to possess a specific influence upon this part, and exerts its influence for the better speedily and effectively. I employ it in combination with *aconite,* using five or eight drops of specific aconite to fifteen or twenty of rhus, in half a glass of water, giving a teaspoonful every hour. Where the tongue is reddened at the tip and edges, and pointed, tremulous on protrusion, or where the patient is particularly restless, or nauseated, it is especially commendable.

Markedly necrotic conditions might suggest the use of *baptisia,* though it would be difficult to imagine a case where baptisia would succeed if echinacea had failed to arrest the tendency to gangrene. *Lachesis* should be borne in mind where phagedenic tendencies are pronounced, two or three grains of the 6x or 10x, every two hours.

Periodicity might be present in a malarious region, and demand the use of an *antiperiodic.* In such a case, the antiperiodic would constitute an important part of the treatment, and it should be used appropriately, the exacerbation being anticipated with proper doses of *quinia sulphas* for several days, until the periodicity has been interrupted. In treating children and delicate persons, the 3x trituration of *arseniate of quinia* may be found more acceptable to the stomach, less disagreeable to the nerves, and fully as effective, given in two- or three-grain doses, four or five times daily.

Local applications should figure extensively, in the treatment of

erysipelas. *Echinacea* is one of the best of these, its antagonism to necrosis of tissue being as well marked locally as constitutionally. Cloths saturated in a twenty-five per cent dilution of a saturated tincture or the specific medicine, in water, should be laid upon the affected area, and renewed every hour. *Acetate of lead* is an application which is soothing and cooling, and was once a favorite local remedy with me. A saturated solution, in water, may be applied on saturated cloths, frequently repeated. Another excellent agent is *citric acid*, used in saturated aqueous solution, as above directed. Another remedy, which many laud very enthusiastically, is the *spirit of Mindererus*. This is probably the best use this old formula can be made of, as its local influence in erysipelas is sometimes remarkably fine.

Cathartics can exert no beneficial effect in the treatment of this disease, and should be avoided, as their use is unscientific and uncalled for. Enemata may be employed where evacuation of the bowels is an urgent matter, though the small amount of food consumed will obviate necessity that the bowels move every day.

Complications should be met by rational measures. Abscesses should be opened early, and cleansed with dilute peroxide of hydrogen, diluted echinacea, or weak solutions of carbolic acid and glycerine, in water. Ophthalmic complications will call for the local application of diluted echinacea, a weak solution of citric acid or sugar of lead. Cardiac complications may demand calcium fluoride, cactus grandiflorus, or convallaria.

The *diet* should be nutritious but not stimulating, fatty meats and high seasoning being forbidden. Milk, plain and malted, farinaceous foods, digestible fruits and vegetables, and eggs, well cooked or rare, will constitute an appropriate regimen.

XVII. SEPTICÆMIA AND PYÆMIA.

Septicæmia and pyæmia are often confounded. Some medical writers have even failed to distinguish between them, but have regarded the two conditions as identical. However, there is at least one distinguishing feature, and that is that pyæmia is marked by the diffusion of abscesses through various parts of the body, as the result of lodgment of emboli distributed from a primary abscess, while septicæmia is a general poisoning of the fluids, without foci of suppuration. Each will be considered separately.

Septicæmia.

Definition.—A general febrile disease, without foci of suppuration, caused by the absorption of septic bacteria and their ptomaïnes—usually bacteria of suppuration.

Etiology.—Septicæmia may result either from the absorption of toxines from without the circulation (septic intoxication), or from the generation of toxines in the circulation, through the multiplication of septic bacteria within the blood-vessels (septic infection). In either case, absorption of septic material or bacteria must first take place from some nidus of putrefaction in intimate association with the circulation, such as a pent-up wound, a retained placenta undergoing decomposition, typhoid ulcers, old tubercular cavities, etc., from which free access of air is excluded. Cavities open to the air hardly ever become the origin of septicæmia.

Symptoms.—There is great variation in the severity of the symptoms of different cases of septicæmia, depending upon the amount and intensity of the septic material giving rise to them. Some cases may be so mild as to be almost overlooked, while others are so profound as to result fatally, within one or two days after the onset.

Senn, in his Principles of Surgery, segregates cases of septicæmia into three general classes: namely, (1) fermentative fever, (2) sapræmia, and (3) progressive septicæmia.

Fermentative fever (resorption fever) is the simplest form of wound complications, the absorption of mildly septic fluids being similar in results to those of transfusion, or the injection of pepsin into the blood. It may follow slight injury or operation, especially operations where superficial necrosis in wounds attends the action of solutions used in dressings; or it may result from extravasation of blood. Soon after the development of the provoking cause—within a few hours—a mild fever (without a chill) arises, the temperature rapidly running up to 103° or 104° F., where it may remain for twenty-four or seventy-two hours, when it subsides spontaneously, no severe constitutional symptoms appearing at any time.

Sapræmia is a form of septic intoxication due to putrefactive changes occurring in dead material. Ptomaïnes are thus formed, various microörganisms being concerned at divers times, such as pyogenic bacteria, and various forms of the proteus group. As these microbes multiply and grow, toxines are developed, which are absorbed into the circulation, and produce the condition just named. Sapræmia, then, is the toxæmia resulting from the introduction of ptomaïnes into the blood from a putrefactive localized focus. Soon after the absorption of such material, constitutional symptoms develop; a slight chill is followed by marked reaction, the temperature rising to 103° or 104° F., with rapid pulse, headache, perhaps nausea and vomiting, and *great prostration.* Typhoid symptoms rapidly follow, there being restlessness and delirium, with reddened,

pointed tongue, which later becomes dry and contracted, or presents a glazed appearance. Three factors are necessary to produce this condition: namely, dead tissue, putrefactive infection of this material with septic microörganisms, and time for the ptomaïnes to be absorbed. A focus of putrefaction may be due to lacerated or bruised tissues, blood-clots in wounds, to retained secundines, etc.

Progressive septicæmia is due to more than absorption from a localized focus of putrefaction. In addition to ptomaïnes absorbed, microörganisms within the blood continue to generate toxines. The microbes most common in this form of septicæmia are the pyogenic bacteria.

The *symptoms* of progressive septicæmia are developed soon after the absorption of the septic material; seldom later than the third day, and often within twenty-four hours. They resemble those of sapræmia, only they are more profound. There is an initiatory chill, followed by a temperature of 103° or 104° F., with varying intermissions. The pulse is weak and wiry from the start, evidencing great prostration, and it soon becomes soft and compressible. Inflammatory action may proceed rapidly along the lymphatics, from the focus of putrefaction to vital organs. The patient inclines to drowsiness and stupor, early, though he may be aroused by violent vomiting alternated with diarrhœa. The face presents a yellowish pallor, and assumes a vacant expression; the pupils are often dilated, and the tongue is dry, and red at the edges, with a brown dorsum. Such cases are liable to prove fatal within from two to four days.

Diagnosis.—The difference between the symptoms of this disease and pyæmia is sufficiently characteristic to enable the practitioner to readily differentiate. In septicæmia there is not the pronounced chill at the initiation that marks pyæmia, which is ushered in by a pronounced rigor. In pyæmia the chills recur, and are as prominent as an ague, in some cases, while in septicæmia there is but the one chilly period, and that is at the onset, and it usually amounts to only slight shivering, or mild rigors. In septicæmia the temperature rises rapidly to 105° or 107° F., while in pyæmia it gradually rises to 102° or 104°. The skin, in pyæmia, presents a peculiar leaden yellow hue, while in septicæmia there is not this peculiar discoloration. Pyæmia develops gradually, while septicæmia is a disease of rapid onset. The history of the case will usually assist in determining between septicæmia and typhus or typhoid fever, should there be any question in this direction.

Prognosis.—The prognosis will depend upon the amount of septic material absorbed in the beginning, and upon the facility with which the putrefactive focus can be evacuated, and rendered aseptic.

Where the symptoms are mild in the beginning and it is possible to evacuate the offending material, as by cleansing the uterus, when it contains putrefactive placental remains, with proper curettage, there is good ground for a favorable prognosis. But, when the onset is violent, the patient being immediately seized with urgent vomiting and purging, delirium speedily following, there is but little prospect that recovery will follow. Collapse and dissolution are liable to soon attend such a condition.

Treatment.—The treatment of severe cases of septicæmia will be more of a surgical than therapeutic nature. Septic cavities should be drained and cleansed with antiseptic solutions as thoroughly as possible, at an early date. If the uterus contain putrefactive material, it should be evacuated by proper curettage and flushed frequently, with warm antiseptic solutions, until constitutional symptoms have passed away. In puerperal peritonitis, where the peritoneal sac contains septic material, the only probable chance for the life of the patient is a thorough cleansing of the cavity with antiseptics, through an abdominal incision. All putrefactive cavities should be repeatedly flushed, until dead material has been completely removed. *Therapeutic measures* may accomplish some good. Tongue indications should be carefully observed, and any prominently indicated dynamical antiseptic administered, as soon as called for. *Sulphite of sodium* or *sulphurous acid* may be required, though *baptisia* or *echinacea* may often serve a good purpose. Professor Scudder's favorite remedy for septicæmia due to putrefying placental material, when there was fetor about the patient, was minute doses of *potassium chlorate.* To counteract the prostration, heart stimulants, such as *nitro-g'ycerine* or *strychnia* (hypodermically), may be demanded to tide the patient over. Restlessness and delirium, with pyrexia, may call for *aconite* and *rhus tox.*, *gelsemium*, or *jaborandi.*

Lachesis, in 2-grain doses of the 6x trituration, repeated every two hours, is an excellent internal remedy to correct the septic condition of the blood.

Prophylaxis consists in observing proper antiseptic precautions in the management of wounds, abortions, and obstetrical cases.

Pyæmia.

Synonym.—Pyothæmia.

Definition.—A general infectious febrile disease, resulting from the entrance of emboli infected with the microbes of suppuration into the circulation, characterized by the formation of metastatic abscesses in various parts.

Etiology.—The cause of pyæmia was once believed to be the

absorption of pus into the circulation from primary suppurating surfaces. Later, Virchow called attention to the part played in the genesis of the metastatic abscesses by thrombi and emboli, and later investigators now declare that these emboli must be charged with pyogenic microbes (infected), in order that infarctions shall degenerate into embolic abscesses. The results of lodgment of non-infectious material (emboli) are simply mechanical—infarctions—but when a thrombus contains pyogenic bacteria, the leucocytes and embryonic cells degenerate into pus-corpuscles, and a focus of suppuration results. When pus-organisms induce coagulation necrosis in the smaller vessels about suppurating wounds, producing thrombi and purulent phlebitis, small fragments of the thrombi (emboli) are carried by the circulation to different parts, where they find lodgment, the pus microbes there forming colonies, and setting up suppuration.

Pathology.—The distribution of metastatic abscesses depends upon the location of the primary distributing focus—on its relation to the special portion of the circulation involved.

In external wounds and osteo-myelitis, as well as in acute cutaneous phlegmon, the embolic abscesses are most liable to develop wedge-shaped infarcts in the lungs; though the emboli may pass through these organs, and become lodged in the liver or kidneys.

When the primary suppurative foci are in the first capillary distribution of the portal circulation, as in the intestines in typhoid fever, the metastatic abscesses appear in the substance of the liver, with or without pyelo-phlebitis.

Ulcerative endocarditis may result in showers of small metastatic abscesses which invade the lungs when the right endocardium is involved, and the spleen, kidneys, intestines, and skin, when the suppurative action is in the left heart (the arterial pyæmia of Wilks).

So-called idiopathic pyæmia occurs, in which the primary lesion is not apparent, but in which numerous abscesses are scattered about, in various parts.

The blood, in pyæmia, tends to spontaneous coagulation in the vessels, wherever there is slowing of the current. Colonies of micrococci are found in various places in the blood, and on the walls of the vessels.

Pyrexial changes are observed in the internal organs and other soft tissues, similar to the granular degeneration marking other febrile diseases. The spleen is swollen, and exhibits parenchymatousdegeneration.

Pyæmic inflammation of the serous membranes is often present, the pleura, peritoneum, and pericardium, being involved. The pleura

is especially susceptible, the pleural cavity sometimes filling rapidly with purulent material. Suppurative arthritis may occur, and lymphangitis is liable to arise in the neighborhood of metastatic abscesses.

Symptoms.—*Chills* are important symptoms of pyæmia, these occurring at the commencement of the disease, six or seven days after the infection which gives rise to it has begun. The chills may recur regularly or irregularly. When they recur with regularity, the condition is liable to be mistaken for malaria. The more frequent the chills, the more numerous the metastatic abscesses, the chilliness usually heralding the origin of a new point of infection, and attending the commencement of suppurative action.

The *fever* which attends varies in character, though it is usually intermittent or remittent. When intermittent, the temperature may rise to 104° during the acme, continue there a few hours, then subside, with sweating, to normal. In some cases there may be several chills during twenty-four hours, with paroxysms of fever between, each chill being preceded by a remission, or the temperature falling to or below normal.

Gastric symptoms are not usually so marked as in septicæmia, nausea and vomiting seldom occurring; and *delirium* is rarely present, unless the brain be the seat of metastatic abscesses, the mental condition continuing sound throughout. The *pulse* soon becomes feeble and rapid, and the skin assumes an icteric tint, due, supposably, to the destruction of red corpuscles and consequent staining of the skin with hematoidin.

The local as well as the general symptoms vary in proportion to the number and location of the abscesses. Where many emboli are diffused throughout the body, they are usually small, and the local symptoms are obscured largely by the severe and rapidly fatal general disturbance, the disease terminating with typhoid symptoms and death, in from one to three weeks. In those cases where the emboli are fewer, a more chronic course follows, and the active constitutional symptoms are less severe. Such cases are more chronic, and the locations of the abscesses are indicated by pain, and varying functional disturbances.

When the lungs are involved, *dyspnœa* will be a constant symptom, its extent being determined by the number and size of the abscesses. Large abscesses located near the pleura will give rise to pleural inflammation, signalized by lancinating pains and dry crepitus, or friction sounds, upon auscultation. Over the region of the infarct may be heard crepitant râles and bronchial respiration, and percussion will now discover dullness. If the abscesses

be located near the heart, the pericardium may be involved, and cardiac symptoms supervene.

Embolic abscesses in the kidneys will be determined by the presence of albumin and pus in the urine. Arthritic abscesses will be easily recognized by the redness, swelling, and pain. Other locations may be involved where the symptoms are obscure; large abscesses may develop internally, so insidiously—without pain—as to reach immense proportions before they are recognized. In other cases, the subcutaneous connective tissue may be involved, with the symptoms of ordinary abscess.

Chronic cases may linger along for months, before a final fatal termination, the patient gradually losing flesh from the constant hectic, until death from exhaustion results, or extensive amyloid degeneration of vital organs interferes with processes necessary to the maintenance of life.

Diagnosis.—The history of the case will usually assist in determining the condition of affairs, unless there be idiopathic pyæmia present where no local focus of infection is known. If malaria be confounded with it, quinine may be employed for diagnostic purposes, it being remembered that while this drug will interrupt the chills of malaria, it exerts no pronounced influence over those of pyæmia. Remembering the points of distribution of emboli, we will hardly be liable to confound this disease with acute atrophy of the liver, acute rheumatism, or typhus, or typhoid fever.

Prognosis.—The prognosis is always unfavorable. However, patients of powerful recuperative energies, who are not severely affected—where the abscesses are few and far between—sometimes recover.

Treatment.—It is doubtful that there are anti-suppurative remedies sufficiently potent to arrest the action of the pyogenic microbes after they have once entered the circulation as extensively as in pyæmia. It may be worth while to attempt this with *echinacea*, in acute cases, and with *calcium sulphide* or *berberis aquifolium* in more chronic ones; but the outlook will not be very promising. However, there is nothing like faith and perseverance, and these remedies judiciously administered can do no harm, at least. The anti-suppurative action of *potassium choride* 3x should also be recollected here.

Abscesses, when accessible, should be treated antiseptically, and stimulants and nutritives should be regularly administered, to sustain the patient as much as possible.

Allopathic authorities advise the free use of *alcoholic* stimulants, it being their belief that life may thus be prolonged, in some cases, until the disease subsides.

XVIII. ASIATIC CHOLERA.

Synonyms.—Epidemic Cholera; Spasmodic Cholera; Malignant Cholera.

Definition.—Epidemic cholera is an acute, specific, infectious disease, endemic in some parts of India, but carried, in epidemic form, to other localities. It manifests itself either by choleraic diarrhœa, having no distinct characteristic; cholerine, which differs but little clinically from cholera-morbus; and pronounced cholera, characterized by copious "rice water" purging, persistent vomiting, severe muscular cramps, marked prostration, emaciation, and collapse, rapidly followed by dissolution, or recovery. The dejections of the several varieties contain the distinctive cholera bacillus.

Historical Note.—Asiatic cholera has prevailed in India for centuries, but until the great epidemic of 1817, very little was heard of it outside of the medical reports of the East India Company. The statement of some authorities that cholera originated at Jessore in 1817 is erroneous, as there were ten extensive epidemics on the Indian peninsula from 1503 to 1817.

The great cholera epidemic of 1817 first attracted general attention, from the extent of territory traversed and the appalling loss of life that followed. Within an area of 195,935 square miles, almost every town and hamlet suffered from its ravages. Europe had thus far escaped; but the epidemic of 1827 did not stop at the boundaries of India, but, advancing through Afghanistan and Persia, it moved on to Russia, and by 1832 it had devastated the whole of continental Europe, and had spread to America. In 1840, during the Opium War, the English troops carried the disease from India to China. From the extreme east of Asia it now began its westward march, and traversing the length of the continent, entered Russia, in 1846. Here, after decimating the Empire, it continued its progress westward, over Europe. As in 1832, it again crossed the Atlantic, but this time did not cease its progress until it reached the Pacific Ocean, having traversed the habitable globe in the space of eight years. In 1851–53, cholera again reached Europe over the old route, *via* Russia, and passed with emigrants to this country, where it prevailed widely, though not so extensively as in the former epidemic.

The outbreak of 1865 chose a new route. Beginning in the Bombay Presidency, it traveled to Mecca, where 30,000 died of the disease. Leaving the Holy City, it passed to Alexandria, *via* Suez, crossed the Mediterranean to Europe, and reached New York in 1866.

The last visitation in this country occurred in 1873, the disease, as before, entering Europe through Russia, and being brought here by European emigrants.

The Egyptian epidemic occurred in 1883, and was, as before, directly traceable to pilgrims. A stringent quarantine prevented its passing to Europe.

The last European epidemic occurred in 1892. It originated in the Punjab, and was rapidly disseminated over India. The great Twelfth Year pilgrimage, with its million pilgrims, was broken up, and flying devotees carried the plague in all directions. It reached Europe inside of six months. America warded it off by a rigid quarantine, and, although New York harbor was full of infected vessels, only two cases occurred on the mainland.

Etiology.—The etiology of cholera has been a prolific cause of controversy, and, although the doctrine of Koch is adopted by the majority of the profession, there is a respectable minority who reject (or only partially accept) his ideas. Numerous theories have been advanced, but the subject is too extensive to permit of their being discussed here.

Koch, in 1884, advanced the idea that the disease was due to a specific microbe, the comma bacillus, which gained entrance to the alimentary canal by contaminated food and water. The bacteria are shorter but more bulky than the tubercle bacilli, and slightly curved; hence the name—comma bacillus. Some are joined, and form an S, and, again, they frequently grow in spirals. Koch himself inclined to the opinion that they were a transition form between bacilli and spirilli, if not genuine spirilli. They are found in the dejections, and in the structure of the intestine. Rarely, they are noticed in the vomitus, but in that case regurgitation through the pylorus is supposed to account for their appearance. They can be cultivated in various media, but drying destroys their vitality. In this they differ from many other bacteria, notably the bacillus tuberculosis.

COMMA BACILLUS OF CHOLERA

Cholera is endemic in certain localities on the Indian peninsula, and within this district the disease is always in existence. Here, peculiar climatic and topographical conditions, an overcrowded population, and the utter disregard of sanitary measures among the natives, furnish an opportunity for the development of microörganisms, scarcely to be equaled elsewhere. There have been no epidemics which cannot be traced back to the Bombay Presidency. The disease always proceeds along the routes of travel and commerce, without regard to climate. Trading caravans, invading armies, and pilgrim hordes, have disseminated the disease, time and again.

Cholera is not contagious; or, like typhoid fever, only exceptionally so, and physicians and nurses handle patients with impunity.

The bacilli may gain admission to the system directly, as from food and water infected from fecal discharges, or they may multiply outside of the body, and, contaminating the water supply, reach the system indirectly. As the disease is propagated from the stools, bad sanitary conditions furnish the opportunity for its spread. Milk, and vegetables washed in water containing the bacilli, are often sources of infection. Soiled garments and bed linen are responsible for a great many cases of cholera. This has been noticed frequently in the cases of washer-women. In the East, the custom of wearing the clothing of the dead is common. It is, however, through a vitiated water supply that cholera is principally propagated, and it is thus enabled to disseminate itself through a community with astonishing rapidity. The disease does not prevail extensively in high altitudes. Hot, sultry weather is favorable to its development, and epidemics are more common in summer and autumn. However, it may be stated that one of the worst Russian epidemics occurred during an exceptionally severe winter. No age is exempt, and the poorly fed, debilitated, and intemperate are especially prone to be attacked.

It is an eastern proverb that fear kills more than cholera, but it is doubtful if the emotions have as much to do with rendering the body susceptible to the disease as some writers imagine. Authorities differ as to one attack conferring immunity against a second. If this is the case, the duration of the period is short.

Pathology.—The post-mortem appearances vary considerably, depending on the stage of the disease in which death resulted. The temperature frequently rises after death, and the body cools slowly. Rigor mortis begins early, and the rigidity is marked, the limbs often being distorted. Post-mortem movements are a peculiar feature, sometimes changing the position of the body. These movements have often given rise to reports of persons being buried alive.

Decomposition is late in making its appearance. The integument has a leaden pallor, and is mottled and wrinkled. The blood is thick, tarry, and slightly coagulable, darker in color, and slightly acid. There is marked dryness of the tissues. The peritoneum is dry, and covered with a viscid substance. The stomach presents no characteristic appearance. The small intestines usually contain a turbid, whey-like fluid and the cholera bacillus. The epithelial denudation is probably post mortem. The mucous membrane is swollen, and usually pale. The solitary and Brunner's glands, and Peyer's patches, are swollen and prominent, the latter congested, and occasionally ulcerated. The large intestine is frequently collapsed,

and the solitary and agminated glands swollen. Cases have occurred where the colon appeared normal.

Symptoms.—The period of *incubation* varies from two to five days, but in exceptional cases only a few hours elapse before the disease manifests itself.

The symptoms vary greatly in severity in different cases, as is characteristic of infectious diseases generally. The degree of intensity does not necessarily depend on the number of bacilli that gain entrance to the body, although this should usually be the case. Predisposition and physical conditions have undoubtedly a great influence in determining the severity of the disease. Although the varieties merge into one another, there are three recognized types that are present during an epidemic, and it will be conducive to an understanding of the disease to describe them separately.

Choleric diarrhœa cannot be differentiated from ordinary diarrhœa, except by bacteriological investigation. A patient may have what he considers a simple diarrhœa and unwittingly communicate the disease to others, without ever knowing that he has had Asiatic cholera. All diarrhœas should therefore be looked upon with suspicion, during an epidemic of cholera. As a rule, the flux occurs suddenly, and the discharges are copious and thin. They range from three to five during the twenty-four hours. Sometimes they are more frequent, and then are not bile-stained. Colic and griping are not usual. There is a coated tongue, slight nausea, headache, and occasionally slight cramps in the legs. The duration of the attack is from several days to two weeks, often tending to relapse, or merging into the more serious forms. The Koch bacilli are present, and can be demonstrated.

In *cholerine*, there is vomiting as well as purging, diarrhœa not necessarily preceding the former. An attack resembles cholera morbus very closely. The stools are larger and more frequent than in choleraic diarrhœa, and soon become serous, resembling the rice-water discharges of pronounced cholera. They contain the cholera bacillus. Emesis soon follows the diarrhœa, and after the contents of the stomach are expelled, the vomitus becomes watery and tasteless. There may be considerable thirst. There is pain in the epigastrium, and abdominal discomfort, but griping is not necessarily present. Cramping of the muscles of the legs is usually noted. The patient complains of being faint and dizzy. The urine becomes scanty, and, if the purging persists, may become suppressed. Albumen is not unlikely to be present, in severe cases. The skin is cold, the voice becomes hoarse, and the features have an anxious expression.

Some cases take on a typhoid condition, with a slight febrile rise,

resulting in a slow recovery. In others, the symptoms are severe, and collapse comes early in the disease. Others, again, develop pronounced cholera, with all its characteristic symptoms.

Recovery from cholerine is the rule, though relapses are frequent, and convalescence slow.

Pronounced Cholera.—Here, we recognize three different stages: the prodromal stage, the stage of attack, and the stage of reaction.

The *prodromal* stage varies greatly in different cases, and is sometimes not apparent, the disease beginning with its more severe manifestations. The period of incubation varies from twelve hours to several days, and there is malaise, more or less depression, headache, restlessness, slight digestive derangement, and a feeling of discomfort, followed, as a rule, by symptoms similar in character to those described under the head of choleraic diarrhœa. Occasionally there is no flux during this stage.

The *diarrhœa*, if it has preceded the period of attack, becomes more severe, and assumes the rice-water appearance, so characteristic of this disease. The number of evacuations increases from four or five to twenty, or, in some cases, sixty a day. There is little or no pain in evacuating the bowel, the act being performed without much effort, the discharges coming away in a stream. Emesis sets in early, and, after the contents of the stomach are evacuated, the vomitus becomes whey-like, resembling the discharges from the bowel. The vomiting is projectile, and not attended with nausea. It is frequently attended with a distressing singultus. This excessive loss of fluid produces a drying of the tissues, and the blood becomes diminished in quantity, and thickened. The secretions are arrested, but the sweat glands increase in activity, and the patient is covered with a clammy perspiration. The cramps in the muscles, especially those in the legs, are extremely painful, but outside of this there is little complaint.

The patient himself often expresses surprise at the little discomfort attending the excessive discharges. Sensation is probably blunted. As the disease progresses, the patient emaciates rapidly, the skin is cold and dusky, the lips blue, features pinched, and eyes sunken. The hands are wrinkled, like those of a washer-woman, the breathing is short and hurried, increasing to thirty, sometimes forty, respirations per minute. The pulse is small and rapid, at times disappearing from the wrist. Although the patient's temperature is subnormal (at times 80° F.), he complains of heat, and resists efforts made to increase the warmth of the body. He is apathetic, and lies motionless and indifferent, although conscious. This condition lasts from three to forty-eight hours, resulting in death, or reaction.

Stage of Reaction.—After passing through the distressing symptoms of the algid stage, the patient may gradually rally. The temperature rises, the pulse and respiration improve, the cramps cease, the stools become infrequent and more fecal, vomiting ceases, the urine is secreted, but is at first scanty, high colored, and albuminous. Convalescence is slow, and relapses common.

Complications.—*Cholera typhoid* is a common complication at this stage, and often carries off the patient already weakened and debilitated by disease.

Uræmia is a serious complication, and frequently fatal. After reaction, the urine still remains suppressed or scanty, and very albuminous. The patient is drowsy, face flushed, pulse slow, and bowels constipated. There is headache, at times delirium. A spinach-like material is vomited. There are convulsions, coma, and death.

Cutaneous eruptions frequently make their appearance in the second week of the attack, during convalescence. All the varieties of the acute exanthamata have been noticed. They appear in a variable percentage of cases, during different epidemics. As low as 1%, and as high as 46% of cases have been noticed. It is regarded as a favorable sign, and is usually followed by an improvement in the patient's condition.

Diphtheritic inflammation of the mucous membrane is a frequent cause of death, the upper air-passages suffering most frequently.

Pleuritis and *pneumonia* are occasional complications. *Suppurative parotitis* is less frequent, but usually results fatally, from pyæmia.

Diagnosis.—The epidemic character of the disease, and its great mortality, should prevent error in diagnosis, although, at the beginning of an epidemic, isolated cases may not be recognized.

Cholera morbus is the only disease with which it can be confounded. We have here vomiting, rice-water discharges, and collapse, as stated under the head of cholerine, the presence or absence of the characteristic bacilli often being the only means of diagnosis between that variety of Asiatic cholera and cholera morbus.

Poisoning by arsenic or antimony differs greatly in its clinical features. The vomiting is painful, and preceded by burning in the stomach and œsophagus. The diarrhœa is not of the rice-water variety, but mucous- and blood-stained. The poison is easily detected by analyzing the vomitus or dejections.

Prognosis.—The prognosis is very unfavorable, as the average mortality is 50%. Epidemics vary in severity, and the death rate ranges between 20% and 80%. The disease is particularly fatal among the aged, young children, and the intemperate. Where cholera develops rapidly, the prognosis is grave, a gradual increase being

considered a favorable sign. The mortality is always greater during the early history of an epidemic, the virulence of the disease seeming to be mitigated during the succeeding months. If the disease does not find favorable conditions for its further development, its intensity is gradually exhausted. The death rate is increased as the equator is approached.

Treatment.—PROPHYLAXIS.—In a disease where one man may disseminate the seeds of an epidemic, preventive measures stand first. Hygienic conditions, both private and public, have an important bearing on the spread of cholera. Cess-pools and privy vaults should be disinfected, and all standing and stagnant water should be drained, if possible. Filth should not be allowed to accumulate. The water supply should receive attention; where there is a possibility of infection, the water should be boiled. Quarantine regulations should be enforced. Of the value of a rigid quarantine, we have had a striking example in the cholera epidemic of 1892, when, at New York, the disease was prevented from gaining an entrance into this country. Cholera stools should be disinfected and buried. Clothing that has been in the sick-room should be thoroughly disinfected, especially when soiled by the patient. Everything that can possibly spread the disease should be cleansed or destroyed.

The *medical treatment* of choleraic *diarrhœa* will be similar to that of ordinary serous diarrhœa. The compound *tincture of cajeput* (American Dispensatory) may be administered in fifteen- or twenty-drop doses, repeated every fifteen or twenty minutes. Instead of this, or in combination with it, a decoction of the fresh *erigeron canadense* plant may be taken freely, the patient being made to drink a wine-glassful every fifteen or twenty minutes, until its action in arresting the evacuations becomes manifest. Or, instead, one may employ three-grain doses of *arsenite of copper* 6x, repeated every half hour, until two or three doses have been taken, then every hour, until relief follows.

In *cholerine*, the first important step will be to arrest the vomiting. This we will probably be able to do with minute doses of *aconite* and *rhus tox.*, as follows: ℞ Specific aconite gtt. v–vii, rhus tox. gtt. x–xv, aqua ʒiv. M., and order a teaspoonful every fifteen minutes, until the vomiting ceases. As the emesis becomes arrested, the intestinal evacuations will usually cease. Where need of stimulants is apparent, we may derive better results from the compound *tincture of cajeput*, used as already advised.

In *pronounced cholera* such remedies must be aided by the application of brisk *cutaneous stimulants*, by means of sinapisms, capsicum liniment, or friction with dry capsicum aided by dry heat. The

hypodermic injection of a third of a grain of *muriate of pilocarpine* will assist in equalizing the circulation and modifying the severity of the symptoms where there is much elevation of temperature; and where algid symptoms are prominent, the thirtieth of a grain of *strychnia* may be employed, the dose to be repeated at the discretion of the practitioner. To relieve the urgent thirst, the combination of *aconite* and *rhus tox.* will be found excellent, and as a drink, a cold decoction of *erigeron canadense* will be best.

The patient must invariably remain in the *recumbent* position, and on no account rise to stool, or for any other purpose. The effort and change of position are almost certain to bring on aggravation of the intestinal disturbance, and repetition may render an otherwise favorable case fatal.

Occasionally, *acids* or *alkalies* may be specifically indicated.

The *diet* is an important consideration, both from a prophylactic and curative standpoint. As cholera is a zymotic disease, the germs may be lurking in any raw food or drink that may be taken, and strict sterilization of everything taken should be observed, and no raw food or drink of any kind allowed. It should be a standard rule to boil everything, and food should not be served in dishes which have not been washed in boiling water. Sterilized water should be used in the preparation of medicines, and for drinking, and rinsing the mouth. As cholera germs do not thrive in acid media, acid beverages may be drunk freely, if they do not seem to aggravate the gastro-intestinal disturbance. As a *prophylactic*, sour lemonade, further acidulated with a few drops of sulphuric acid, has been highly recommended. Vinegar, lime juice, and other sour drinks may be employed for the same purpose.

As alkaline fermentation in the stomach favors infection, only plain food should be eaten during the prevalence of an epidemic, pastries and fried dishes being avoided.

Lastly, during a cholera epidemic, it is best to avoid everything likely to ordinarily produce diarrhœa. During the active period of the disease, there is little use of attempting to administer nourishment to the patient, as the diarrhœa and intestinal evacuations are thereby only increased. Now, hypodermic injections of warm salt water (a teaspoonful to a pint of boiled water) should be freely made, into the thighs and abdomen, to replace the drain upon the blood caused by the serous diarrhœa.

When vomiting ceases, and the symptoms of collapse abate, small quantities of fluid nourishment may be gradually and cautiously administered. A teaspoonful or two of pancreatinised milk, koumiss, beef tea, or fresh beef juice, may at first be given. If this be

retained, a little more may be administered after a brief interval, the quantities being gradually increased. Horlick's milk may now be given in small quantities. As the stomach remains feeble and sensitive for a long time, the amount and quality of the food must be gradually advanced to milk, egg albumen in brandy, or champagne, and other nourishing fluid food, before solid food is allowed.

XIX. YELLOW FEVER.

Synonyms.—Febris Flava; Typhus Icterodes; Black Vomit; Yellow Jack. Spanish, Vomito Nigro.

Definition.—An infectious, contagious disease, characterized by sudden invasion, and fastigium of from two to seven days' duration, with termination by lysis, the fall of temperature being attended by a remarkable slowing of the pulse, this decline being followed by a second rise in temperature, attended by phenomenal icterus, hematemesis, albuminuria, suppression of urine, and rapid and profound prostration.

Etiology.—This disease seems to be indigenous to the eastern sea-coast of tropical America, especially the West Indies, where it prevails endemically throughout the seasons, and occasionally spreads as an epidemic, during periods of remarkable territorial receptivity, along lines of travel, into the temperate zones. It is a disease of hot climate, filth, moisture, and a low altitude, the seaports of the tropical regions being its principal places of resort, though at the present time railroads offer ready means of transportation into the interior, during severe epidemics.

The principle of infection is believed to be a microörganism, though all attempts to isolate it have thus far proven futile. According to Dr. John Guitéras (Keating's Cyclopædia) the disease is mild in children in its native haunts—the West Indies—being often unrecognized by diagnosticians, and as common as measles is among children in our own community, nearly all adults in these communities being protected by a previous attack during childhood. Colored races seem especially prone to mild attacks of it while young, and are therefore protected from a second attack in later life, rendering it a notorious fact that colored races enjoy a marked immu-

STREPTOCOCCUS FROM VOMIT (NOT BLACK) OF YELLOW FEVER.

nity. Whether colored people born and bred in the North enjoy any more immunity than white persons, may be a matter of question. Like measles, it seems that an attack in adult life is much more severe than in childhood, and when adults who do not enjoy the advantage of protection come in contact with it, it is remarkably virulent and fatal. It is said that Creoles are less liable to the disease than whites, negroes less than Creoles, and Indians of tropical regions least of all. That yellow fever is not a malarious disease is abundantly proven by the fact that malarial fever is never contracted upon the high seas, it being distinctively telluric in origin, while yellow fever may be spread on shipboard, provided fomites have been taken on in port, and decimate the whole crew, as well as contaminate those from other vessels who may chance to go on board, for any protracted time.

It is asserted by some that yellow fever is not directly contagious from one person to another, but that it is conveyed by fomites, the contagium seeming to accumulate infective power as it is nurtured by confinement and other favoring causes, such as decomposition, warmth, and moisture. It is certain that plenty of fresh air is about as reliable a preventive of the disease (save strict quarantine) as any that has been tried, and it would therefore seem that the infection gains virulency after leaving a subject, through such causes. Severe epidemics in temperate regions are usually checked by the first frosts, and a pronounced "freeze" stamps it out at once.

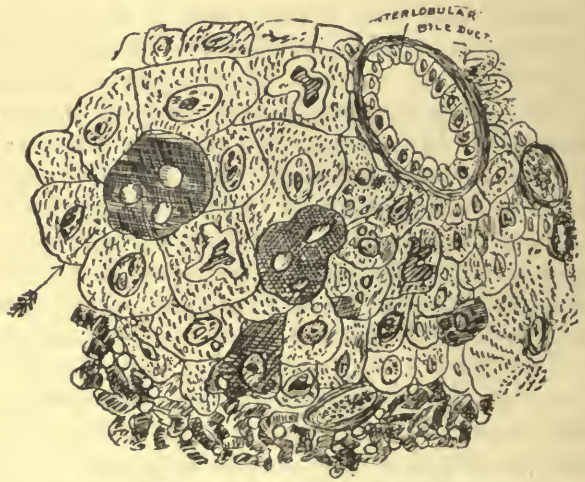
Occasionally the disease invades places far north of its native habitat, a severe epidemic having occurred in Philadelphia, in 1793, four thousand, out of eleven thousand persons attacked, perishing. It first appeared in the United States in Boston, in 1693, and has since appeared occasionally at other points, sometimes in severe epidemics, up to the present time. It has been conveyed as far north as Portsmouth, New Hampshire. Occasionally the infection is wintered over, and appears the following season with unabated severity, as occurred at Memphis in 1879, when it hibernated. During late epidemics, there has been a tendency for the disease to follow lines of railway into the interior from southern seaports, severe and fatal cases occurring far inland.

Pathology.—The anatomical changes of yellow fever are found in all parts of the body, though the most marked disorganization occurs in the *liver*. This organ is found to be markedly yellow in appearance (resembling box-wood in color). Disorganization of the parenchyma has occurred, necrotic masses being found in and between the cells, with fatty degeneration, resulting in the distension of these bodies with oil. In other cases, the degeneration is

granular, the nuclei of the hepatic cells being obscured or entirely destroyed. The organ breaks down on firm pressure, and on section the tissues are found drier than normal, less blood than usual being in the vessels.

The *skin* is markedly yellow, the color varying from dark orange to a bright golden yellow, and petechiæ, eruptions, pustules, ecchymoses, and extravasations are liable to be found upon the surface. The *mucous membranes* may also present a yellowish tinge. The adipose tissue, too, is deeply stained an icteric hue.

Important changes are found to have taken place in the *blood*, to these the marked yellowness of the tissues being due. The red corpuscles are broken down in many instances, or they are serrated and shriveled. The broken-up contents are altered, and the hematin is



LIVER CELLS IN YELLOW FEVER Showing NECROTIC MASSES BETWEEN.-FROM SPECIMEN-

converted into bile pigment, this staining the tissues the characteristic yellow. Ammoniacal decomposition sets in soon after the withdrawal of the blood from the body, due to alteration in its saline constituents, and it is found that there is partial loss of coagulating quality, the fibrin-factors having apparently lost their function.

There is active *catarrh* of the mucous membrane of the upper portion of the alimentary canal, with ecchymosis and varicosis of the superficial veins, the extravasations being largely due to the forcible vomiting of the disease. The urinary tract also affords evidence of similar changes, the kidneys being the seat of parenchymatous inflammation, with fatty degeneration and breaking down of tissue. Infarctions are found in the lungs, pulmonary apoplexy sometimes

occurring. Pleural ecchymosis, with effusion of bloody serum into the pleural sac, is also one of the occasional occurrences.

The *muscles* are darker than normal, their dark color contrasting markedly with the yellow color of the skin and adipose tissues. Marked granular degeneration is found to have taken place in the histological elements, this probably being due rather to some specific poison (ptomaïne) generated during the disease than to simple pyrexial changes, as the temperature in yellow fever is not high enough, nor sufficiently prolonged, to account for the very decided morbid alterations.

The *heart* is soft, friable, and flabby, and the muscle is found to have undergone more or less granular degeneration. The cavities contain considerable broken-down, fluid blood, with occasional clots. Guitéras remarks that he has always found the left heart contracted. Neither the endocardium nor pericardium bear evidence of inflammatory action.

The *spleen* is not prominently altered, though it may be slightly congested, softer, more friable than natural, and of darker color.

Slight changes may be noticed in the *brain* and *spinal cord*. These may consist of hyperæmia (not marked), punctate extravasations in the meninges, and occasionally, effusions in the lumbar and sacral regions.

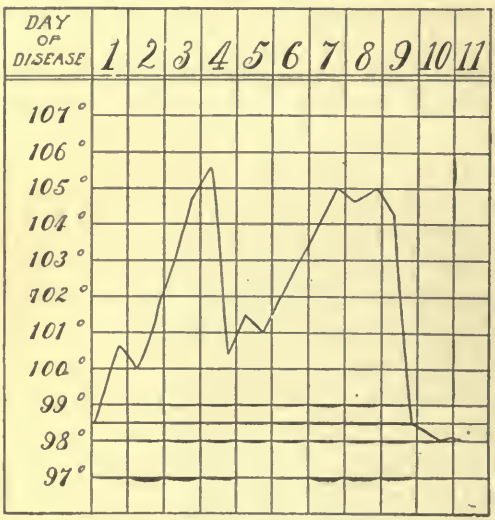
Symptoms.—The stage of *incubation* lasts from a few days to a week. It may, in exceptional cases, extend to fourteen days, but they are rare where the period after exposure extends beyond a week. In cases where but a short time elapses after exposure, the disease usually proves very virulent.

There is a marked difference between the course of a mild attack of yellow fever and a severe one. In the mild attack, the severest symptoms arise during the initial fever, the remission being followed by but slight febrile symptoms, and often these are absent. In all probability the secondary fever is the result of the poisoning of the system by the ptomaïnes generated during the first attack, by the germs of infection which excited the primary fever.

The *invasion* is usually sudden, consisting of a marked chill, speedily followed by fever, attended by severe pains in the head, back, and limbs, the pain being notably severe in the back and legs; and there is nausea and vomiting. The temperature rises rapidly at first, though yellow fever is not ordinarily a disease of very great pyrexia. The pulse becomes full, strong, and rapid at first, though it soon weakens, the skin being dry (sometimes moist) and hot, and secretion arrested generally. The eyes early present a peculiar shining appearance, being markedly suffused, and this, with the bronzed

or yellowed color of the countenance, and staring look, impart to the visage a remarkably sodden and dejected aspect. The *tongue* is covered with a thick white fur from the outset, except at the tip and edges, which are red and bare. The bowels are usually constipated, though hemorrhagic diarrhœa may be present. The mind is usually clear to the last, though delirium may set in in the late stage, the patient being wild and restless, and determined to get out of bed.

By the third or fourth day of the disease, the *temperature* will have reached its height, the thermometer registering hardly ever more than 104° to 105° F., though it may rise as high as 110°. When the fourth day has been reached, the temperature declines rapidly, running down to near the normal line, marking a distinct remission, which may last for two or three hours, or two or three days. This constitutes the period of *calm*, the patient being free from suffering, except that of marked prostration, after which a second rise in temperature begins. This usually begins without a chill, and rises more gradually than during the invasion. The temperature may now again reach 104° or 105°, where it remains a day or two, to fall again, continuing to do so, in favorable cases, until the normal point is reached.



Temperature Curve in Yellow Fever.

The *pulse* of yellow fever, after the first few days, is said to be peculiar, a sensation being imparted to the finger as though there were gas in the arteries, the name "gaseous pulse" being applied to it. It is now markedly compressible. It hardly ranges above 110 per minute, during the fever, and, in mild cases, it may not be more than four or five beats above the normal rate. During the remission, or stage of calm, it falls remarkably, lowering to a thirty or forty rate per minute.

About the third day the *icterus* begins to appear, being observable at first in the sclera, and rapidly spreading over the body. The color is deep, like that of pyæmia, almost bronzed in appearance. It is due to staining of the skin from the pigment formed from the elements of broken-down blood, which are deposited in the tissues, and not from hepatic secretions. The perspiration now stains the

linen yellow, and a cadaveric odor emanates from the body. When the third stage is reached, the color becomes a mahogany hue.

The *vomiting* is a marked feature of the disease, it beginning immediately after the chill, and persisting during both febrile stages. It may be severe or not, in many mild cases being restricted to the rejection of food, as soon as taken. In other cases, the contents of the stomach are ejected first, then a yellowish-green material, of alkaline reaction, is projected, the alkalinity being due to ammoniacal decomposition of the blood. If the case is severe and ominous, there is soon hemorrhage into the stomach, and black contents are ejected, constituting the characteristic "black vomit" of this disease. If, instead of this, yellowish-green, alkaline material continue to be ejected, the symptoms may be considered more favorable.

The *urine* is scanty early in the disease, and, as the morbid changes progress in the kidneys, traces of albumin may be found. It is acid in reaction at first, but as ammoniacal decomposition sets in, it becomes alkaline, and colored with bile-pigment, which becomes more abundant as the disease progresses. If not present before, the bile pigment makes its appearance during the stage of remission, and soon fatty casts, leucin, and tyrosin will appear in the secretion. A strong urinous odor now pervades the entire body. *Complete suppression* of urine will appear in fatal cases, and this symptom may be regarded as an almost certain percursor of a fatal issue, it being asserted by good authority that, though cases with black vomit may recover, those with suppression of urine are almost certain not to.

Diagnosis.—In severe cases occurring during an epidemic, there can hardly be any danger of mistake in diagnosis. The severe attack with vomiting, the peculiar suffused and staring expression of the eye, the marked icterus, coming on the third day, the early decline of the fever in the first stage, with the remarkable slowing of the pulse, and its peculiar compressibility (gaseous pulse), the black vomit, the mahogany color in the last stage, are not all found in other forms of infectious fevers. The history of the case will furnish sufficient testimony in the early part of most attacks to enable the physician to be on his guard against a careless diagnosis. Dr. Guitéras asserts that endemic cases occurring among children are not so easily recognized. Many of the distinctive symptoms are not prominent here, and it may be mistaken for ephemeral fever due to heat, unless considerable care is observed.

Prognosis.—Some epidemics are extremely fatal, others not so markedly so. The epidemic influence (territorial receptivity) will determine, to great extent, the amount of mortality to be expected. One fact is always to be recollected, viz., the disease is ever to be

dreaded when wandering from its indigenous haunts, or when affecting adults who have not been protected by a former attack, especially the unacclimated. Where the yellowish-green vomit continues throughout the disease, the case may ordinarily be considered a favorable one, and when this is replaced by the black vomit, it is always grave, though recovery may ensue. Marked scantiness of the urine is an unfavorable symptom, and when this secretion becomes suppressed, there can be scarcely any hope of a favorable termination. A gradual subsidence of the vomiting, and diminution of the amount of albumin in the urine, may be considered as favorable signs. Convalescence is remarkably slow in this disease, six months being required to thoroughly recuperate from it.

Treatment.—*Prophylaxis* is first to be considered, and this can best be assured by strict quarantine. Not only all individuals who have been exposed or who may be affected, as well as all articles of clothing that have been about the disease, should be strictly excluded from contact with those who have not been infected, but everything surrounding the disease should be strictly avoided by the well who do not possess immunity. Pure, cool air, in elevated regions, is also a good prophylactic, as it will be remembered that the disease is one of filth, warmth, and decomposition. Camping out is practiced much upon these principles in yellow fever regions, when the disease prevails as an epidemic. Even here, however, a strict quarantine should be practiced.

The experience of Eclectic practitioners with this disease seems to have been very limited. Goss states that the treatment should consist largely of the use of antiseptics, and recommends *baptisia* as a leading remedy. *Aconite* for the fever, *belladonna*, when meningeal irritation is indicated, minute doses of *arsenic* for the vomiting, and *camphor*, during the stage of collapse, are the other principal remedies suggested by this author.

The disease is evidently one where *necrotic* changes are at the foundation of the serious mischief which results. The breaking down of the blood, the destructive changes in the liver and kidneys, the black vomit, these are all due to a necrotic tendency, and suggest the most reliable remedy obtainable to arrest it. If we can find a remedy which possesses sedative properties combined with those of an antiseptic, anti-necrotic, and stimulant, we shall have the very remedy required. It will be recollected that the second stage of this disease is attended by a very slow and feeble pulse, and there is therefore a decided objection to any sedative which does not possess, at the same time, stimulating properties. I think that we possess one remedy which is well adapted, in its properties, to the com-

bination of requirements suggested by the pathology of this disease, and this is *echinacea.* I do not write from any experimental knowledge of the disease, for I have never seen a case; but I observe that those who have written from experience seem to be sadly at sea, concerning its medication, and believe this remedy alone would accomplish more than most of the routine treatment usually prescribed.

Baptisia also exerts an influence of this kind, though it is slow in action, when compared with echinacea. The two remedies might probably be used together with profit, the combination being administered perseveringly, throughout the treatment.

Two remedies, exerting a similar influence, and highly prized by the homeopaths, are *lachesis* and *crotalus hor.* Crotalus hor. is especially indicated where a strong hemorrhagic tendency is suggested by ecchymoses, extravasations, and black vomit. Dr. Holcombe, of New Orleans (as well as others), has used it in such cases with excellent success. The 6x of lachesis, and the 3x of crotalus hor. may be employed, both being obtainable at almost any homeopathic pharmacy.

The condition of the tongue early in the disease, as well as the vomiting, would suggest *rhus tox.*, and this might be combined with minute doses of *aconite.* Where the vomiting prevents the retention of remedies per stomach, hypodermic injections of specific echinacea would be philosophical treatment, and it could do no harm, at least.

Scudder recommends the use of an *emetic* where the tongue is heavily coated, and this might be advisable if there were not too much gastric irritation in the beginning. In this disease, however, the morbid condition lies far beyond the reach of remedies which exert a local influence upon the stomach. The blood-corpuscles first, and later, the tissues of important vital organs, become necrotic, and unless we can find a remedy which will neutralize this tendency early, treatment can be of but little avail, in severe cases.

The hypodermic use of *pilocarpine* (1-3d grain) may be resorted to where pyrexial action is dangerously high, and strychnia (1-30th grain) may be administered in the same manner, when a powerful stimulant is required.

There may be malarial complication, demanding treatment for this phase of the disease, but yellow fever is not a malarial disease of itself, as it arises and prevails where no malaria is present. However, *arseniate of quinia* 3x, or *sulphate of quinia,* may be required during convalescence.

Cathartics should be avoided, the bowels being evacuated with enemata, when this is necessary.

The *diet* should be in digestible and nutritious form. This will comprehend the use of such articles as milk, animal broths, pancreatinized milk, Horlick's malted milk, etc. No solid food should be taken for several weeks after convalescence has begun, and fruits and vegetables should be avoided during this time.

XX. MALARIAL FEVER.

Synonyms.—Marsh Fever; Swamp Fever; Paludal Fever.

Definition.—A specific, non-contagious disease, caused by the hematazoa of Laveran, and characterized by periodical paroxysms of fever, tendency to enlargement of the spleen, with general congestion of the portal system, and progressive anæmia.

Etiology.—From almost time immemorial, the origin of malarial fever has been ascribed to the presence of decaying vegetation; and moisture and warmth being necessary to vegetable decay, and such surroundings being the localities where malarial fevers are most prevalent, the natural inference has been that such influence was responsible for the condition of the system which gave rise to them. It was thus widely believed that gaseous emanations, arising from such material, constituted the materies morbi.

But the microscope has enlightened us upon this subject, and it is now known that the blood of a person suffering from malaria invariably contains some form of a species of hematozoa (the plasmodium of Laveran)—a living creature, which undergoes various processes of development in the blood, through which the red corpuscles are destroyed and anæmic conditions brought about, the various phenomena of fever attending, as the results of the presence of this parasite.

Knowledge of other parasitic animal forms which maintain an existence in the blood, assists us in drawing philosophical deductions as to the cause of a disease, upon the nature of which medical men have differed for centuries.

The life history of the filaria is interesting, in illustrating the instrumentality of intermediate influences sometimes concerned in the transmission of infectious diseases, and it is also suggestive of the manner in which the plasmodium malariæ—which belongs to the same family—may be transmitted.

The filaria nocturna, which inhabits the human circulation, in hot countries, is taken up by the mosquito (from the human circulation), during its blood-sucking process, and afterward, when the insect flies away to the water and dies, after gorging itself, is released from the decaying body into the water, to be afterward taken, with

drinking water, into the human stomach alive, and able to enter the circulation of the new host, there to undergo reproduction. Such being the facts with regard to one species of hematozoa, there is great probability that a similar method of transmission with another may be possible.

There are some facts that give such a proposition color, as regards the plasmodium malariæ. Among them may be mentioned that out of many families residing in malarious neighborhoods, it is known that most of those escape malaria, during its prevalence, who confine themselves strictly to boiled water, for potable purposes. Another is, that altitudes and latitudes where the mosquito does not exist are largely free from malaria, while such regions as favor its existence most are the ones where malaria prevails most extensively.

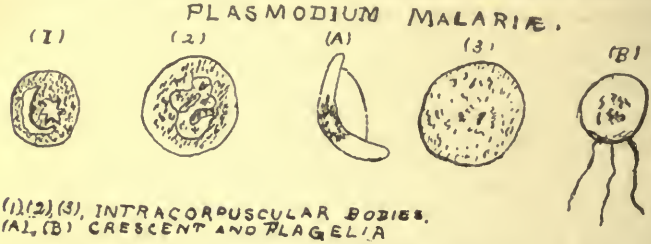
The author finds the foregoing views, penned several years ago (1895, not published, but presented to his class in the winter of 1895–96), have been put forward by Dr. Manson, March 14, 1896, as published in the *British Medical Journal*. It is certainly a very rational view of the matter.

At Dr. Manson's request, Surgeon-Major Ross performed several experiments to test this theory.

Upon placing a person who was affected by malaria, and in whose blood the plasmodium was demonstrable, under a net with mosquitoes, raised from the eggs, and confined, so they could not obtain food elsewhere, and allowing them to suck his blood, the mosquitoes being afterward killed and their stomachs examined the result was that the hematozoa of malaria were found there in plentiful numbers, undigested and alive, and nearly all of them proceeded to develop rapidly into mature forms. Some of these mosquitoes were allowed to deposit their eggs in water, where they died, and this water was afterward drank by a native. Within eleven days afterward, the native developed fever, and his blood was found to contain organisms. The second time, however, the experiment proved without result. We must regard this, then, as only a theory, to be substantiated or disproven in the future. Against it, we have the positive assertions of reliable physicians that malaria has been known by them to prevail extensively in mountainous regions, where the mosquito was never (or hardly ever) seen.

The *plasmodium malariæ* (hematozoon of Laveran) appears in the blood in a variety of forms, these probably representing different stages of development, though it is likely that there are several varieties of malaria-producing organisms belonging to one family but of different species. Their development is as follows:

The *spore*, floating in the blood, attaches itself to a red corpuscle, and finally penetrates it, to absorb, in this situation, nourishment from the corpuscle, and grow to mature form, pigment granules, due to the hemaglobin absorbed from the blood-corpuscle, accumulating in its center. Segmentation of the nematozoon now succeeds, and the spores are set free (by the destruction of the red corpuscle), to attack other corpuscles in their turn. *Crescentric* bodies form, representing one phase of these parasites, and it is believed by some that they are interchangeable into spheres—the fully developed plasmodium or hematozoon. Some believe that the crescents are associated chiefly with irregular forms of malarial fever, and malarial cachexia. Flagella, or lash-like processes, develop from hematozoa which are fully matured.



(1)(2)(3). INTRACORPUSCULAR BODIES.
(A), (B) CRESCENT AND FLAGELLA

Golgi, who leads the Italian school of bacteriologists, believes that the *different forms* of malarial fever depend upon different varieties of hematozoa; that the tertian form depends upon a variety that completes its development in forty-eight hours, the quartan upon one which develops within seventy-two hours, etc. Other types are supposed to be due to the maturity of two or more generations of the same variety occurring at different periods; as, for instance, quotidian ague representing the maturity of two generations of the tertian variety alternately. Probably, also, there may be a mixture of varieties to still more complicate matters, and embarrass regularity of paroxysms.

Differentiation may be made between the tertian and quartan varieties during the stage of sporulation. Then, the spores are found to be more numerous in the tertian variety than in the quartan, while in the latter they are larger.

Malignant forms of malarial fever are also to be differentiated from others by the microörganisms. Not only are the hematozoa smaller, but the spores are also smaller, and less numerous, while the corpuscles shrivel, when attacked.

It is believed that at the time of maturation of the parasite and the liberation of spores, a toxine is set free, which originates the paroxysm, the variety of parasites present (or certain combinations of varieties) determining whether the paroxysm be quotidian, tertian, quartan, etc. When a patient removes from malarious surroundings, the *persistency* of the disease depends upon the presence

of the ehmatozoa in the blood, certain forms probably being more permanent than others.

Conditions Which Predispose to Attacks of Malaria.—*Temperature* exerts an important influence. An average temperature of 58° is necessary for its development, and it does not prevail epidemically short of an average temperature of 60° F. *Moisture* is another essential, and the regions where it is most prevalent are those about marshy districts, where, during the heated season, the water becomes low and stagnant. Salt water is not malarious, but marshes of salt and fresh water combined—as where rivers empty into salt marshes—are liable to be very much so. *New soil*, freshly exposed to the atmosphere, as when prairies have been turned up by the plow, or extensive excavations are being carried on during the building of railroads, canals, etc., is very liable to provoke epidemics of the disease. Regions where there is a non-porous sub-soil, are usually malarious, as the wells are shallow, and the drinking water comes from near the surface. Vegetable decomposition has been supposed to figure extensively in the propagation of malaria, but it is highly probable that the presence of a large amount of such material may be coincident with other conditions upon which the infection actually depends. Extensive irrigation with fresh water under warm sunshine, continued day after day, or often enough to keep the ground moist, is almost certain to be attended by the appearance of malarious diseases in the neighborhood. The infection may be wafted by the *wind* for several miles, when conditions are favorable. The following incident suggests what might be expected under similar circumstances: The crew of a ship which anchored within four and a half miles of a malarious shore, and remained there for several days, were finally attacked by malaria, six days after the wind had blown off the shore for a short time. None of the crew were ailing when the anchorage was made, and all were well until now. As malaria is a disease of the land, never originating on the high seas, the evidence that the disease arose in this instance through the agency of the wind, seems conclusive. Night air is almost certain to provoke aggravation of ordinary malarious infection, upon repeated exposure to it.

Conditions Which Oppose Malarious Infection.—*Latitude* north of 63° north, and south of 57° south, is usually exempt from malaria, though this does not apply to the Pacific Coast, where the Japan current causes a much higher average temperature, far north, along the coast, than usually exists outside of its influence. *Altitude* is another bar to its invasion, an elevation of 1,000 feet being usually exempt from its influence, though mountainous regions, where stag-

nant water is evaporating, under the average temperature essential to its development, may not be free from it. It has been proven that the use of *boiled water* for potable purposes will exempt a large majority of those confining themselves to it during malarial epidemics, though it is doubtless true that the infection may be inhaled, as well as taken with water. *Drainage* is an important factor in the removal of malarious elements from a neighborhood. Many parts of the United States which were once markedly malarious, are now tolerably free from such influence, since the surface has been provided with proper drainage to prevent the stagnation of water upon or near the surface. *Freezing* arrests the activity of malaria germs, though it may not arrest their action when once within the system. However, malarious diseases begin to subside as soon as the autumnal frosts appear, and, though periodicity may be an element in winter diseases, among those who have been previously affected, no pronounced malarious attacks occur in new subjects until the following spring. Large cities, where the ground is thickly set with buildings, and the streets are covered with pavements, are usually exempt from malaria. But, where the city is largely one of residences, and much irrigation of lawns goes on during the summer months, as in California, during the dry season, malaria prevails extensively. In Oakland, California, where there are so many large lawns under irrigation, the summer months are marked by malarious disturbances, though few outbreaks of ague are known, probably on account of the modifying influence of the sea-breeze, wafted through the Golden Gate. *Marine air* neutralizes the propagation of malarial germs considerably. A long-continued sea voyage is nearly a certain cure for malaria, if other means prove futile.

General Pathology.—The morbid conditions which occur in malaria, arise largely from the destructive action of the parasites upon the red blood-corpuscles; for, though there may be a high fever during the paroxysms, the tissue-changes usually due to pyrexial action are not so marked as in more continued fevers, on account of the periods of recuperation afforded here by the intermissions or remissions. However, in pernicious malarial fevers, the extreme hyperpyrexia may lead to early fatal results.

From the development and destructive action of the spores within the corpuscles, we have a large amount of pigment material (hemoglobin) liberated, which becomes distributed through the serum and tissues, and permanently deposited in many of the solid structures. Even in mild malarial attacks, permanent pigmentation of spots in the skin is common, due to the deposit of hematoidin in the rete mucosum; and internal parts are found, upon post-mortem

examination, to afford evidence of a similar abnormal staining. Thus the spleen, liver, kidneys, peritoneum, brain, and other parts may be found to contain deposits of this pigmentary material, derived from the coloring normally held in the red corpuscles, but liberated by the destructive action of the hematozoa. The white corpuscles also become loaded with this material, and are doubtless instrumental in distributing it to various solid structures. The extent of pigmentation varies with the duration and severity of the disease; acute attacks, when not frequently repeated, may not leave much evidence of this character, while in chronic malarial poisoning (malarial cachexia) the staining may be a marked feature.

The destruction of red corpuscles may be followed by two classes of results. In one class, we find disturbance of the spleen and its associate viscera, and in the other those which attend upon impoverishment of the blood from removal of red corpuscles—anæmia and hydræmia.

The *spleen*, being intimately associated with the birth of red blood-corpuscles and the destruction of old ones, seems remarkably disturbed by the abnormal destruction which goes on through the action of the malarial parasite. Simple hyperæmia probably attends at first, especially during the paroxysms, and now there is little structural change, the temporary distention probably only serving to relax and debilitate the tissues of the organ, as autopsies, after death from pernicious malarial fever, demonstrate the spleen to be swollen, soft, and pulpy. But a long-continued and oft-repeated influence of this character is followed by structural changes, due, apparently, to chronic inflammation, as there is abundant evidence of hyperplasia from extensive proliferation of connective-tissue cells. There is enormous enlargement of the organ, and its tissues are firm and resisting. When cut, the capsule is found thickened, and the internal structure is fibrous, and resisting to the knife. Rich pigmentary deposits are found scattered through its substance, and, where the changes have gone far forward, points of melanotic deposit, or amyloid degeneration, may be found distributed throughout the organ. The liver, and other organs connected with the portal circulation, partake, to more or less extent, of these changes.

The loss of red corpuscles entails a condition of hydræmia (the serum being tinged a more or less pronounced chocolate color), and general impoverishment of the blood and tissues. The tissues are pallid and feeble, the circulation being impaired, respiration being hurried upon exertion, and palpitation of the heart arising from slight effort. In advanced malarial cachexia, œdema is a common condition of the tissues, and effusion into the serous cavities is com-

monly found after death. The poverty of the blood conduces to various degenerative changes, amyloid degeneration of different organs occurring in extreme cases of malarial cachexia. When death occurs, it is usually either from exhaustion, or hemorrhage.

In pernicious malarial fever, the ravages of the hematozoa are particularly noticeable, under microscopical examination. The red corpuscles exhibit the presence of the parasite in all stages of development, with the corpuscles in every stage of destruction. The arterioles and capillaries of the brain are crowded, in some places, with parasites, débris of broken-down corpuscles, and pigmented leucocytes.

The following forms of malarial fever are usually described:

Intermittent Fever.

Synonyms.—Fever and Ague; Chills and Fever; Ague.

Definition.—A form of malarial fever, marked by separate paroxysms, each consisting of a chill followed by fever terminating in a sweating stage, with a distinct intermission (return to normal temperature) before the following paroxysm.

Etiology.—Intermittent fever is one of the phases of malarial disease, and is a common form resulting from malarial infection. Whatever tends to depress the physical or mental powers, lessens the ability of the individual to resist the invasion of the infection, and delicate and debilitated persons are usually first to be affected, when a community is invaded by the morbid influence. Intemperance, exposure to the night air, overwork, exposure to chilling draughts of air and other vicissitudes, are among the predisposing causes.

Pathology.—The lesions of this disease are not very marked, and are confined almost entirely to congestion of the internal organs. The *spleen* and *liver* are nearly always enlarged to a greater or less degree, but this enlargement is the result of simple hyperæmia, instead of structural change. These only appear after the paroxysms have been often repeated—after the malarial poison had been influencing the system for a long time. Other internal organs, notably the kidneys and mucous membrane of the intestines, are involved in the hyperæmic condition, though not to so great an extent as the liver and spleen.

The *blood* changes are not so marked as in typhoid, typhus, and some other forms of infectious fever. It clots imperfectly, however, and is abnormally dark in color. Diminution of the fibrin-elements and red corpuscles occurs when the disease continues long, and dur-

ing a paroxysm there is a notable increase in the number of white corpuscles.

Symptoms.—These will depend upon the type which the disease assumes. In the quotidian type, a chill occurs every twenty-four hours, in the tertian type every forty-eight hours, in the quartan type every seventy-two hours. Many cases tend to recur every seven days, and this tendency should be borne in mind during treatment, that provision be made for it. These types may occur in double form, two paroxysms occurring daily in double quotidian, one every day in double tertian, but at a different hour on every alternate day, the paroxysms also differing in character, being marked by severe chill and light fever one day and light chill and severe fever another, or varying in some other particular.

The *paroxysms* are marked by three stages: a cold stage or chill; a hot stage, or the stage of fastigium; and a stage of decline, or the sweating stage.

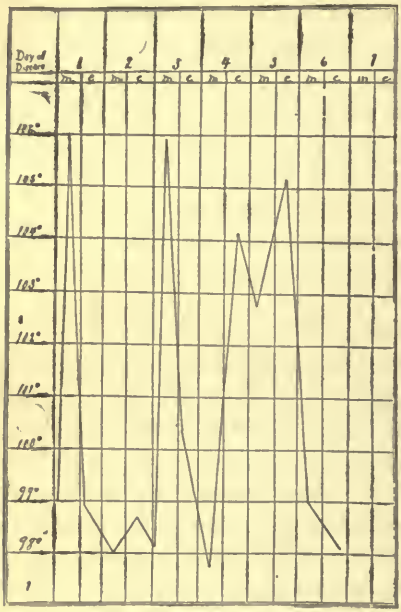
The *cold* stage is characterized by a pronounced rigor; the subject *shakes*, from head to foot. Cold sensations first creep along the spine, but later pervade the entire body. The finger-tips and nose become blue, the skin shriveled, and covered with prominent papillæ (cutis anserina); the face becomes pale, the eyes sunken and anxious, and the voice faint and husky. The sufferer becomes weak and tremulous, and, as the chill continues, he shakes and shivers convulsively, and his teeth chatter. The respirations are now short and sighing; the surface of the body is cold to the touch; micturition is frequent, the urine being pale and limpid. After half an hour or more these symptoms gradually subside; the patient no longer complains of being chilly, and becomes more comfortable.

But he soon realizes that a sense of warmth is rapidly pervading his body, and finds that the warmth is more than that of comfort; the skin becomes dry and hot, the face flushed, the eyes suffused and bright; the pulse bounds, the carotids throb; the tongue is dry, and there is intense thirst, and often vomiting. These symptoms continue to increase in severity until the patient is extremely restless and uncomfortable, the paroxysms often being attended by excruciating muscular pain, this frequently involving the pericranial muscles. During a quotidian ague, the *hot* stage may continue for eight or ten hours, and that of a quartan four or six, though in any case it may terminate in one or two hours.

There is hardly any state where the febrile symptoms are more marked for a short time than in this disease. Though the *temperature* may rise to 104° F. during the chill, it may reach 107° during the hot stage. There is marked arrest of secretion, the urine is

scanty—almost suppressed—and high colored. The skin and mouth are dry, there is intense thirst, the breath is hot, and the bowels are constipated. Severe headache and restlessness often mark this period. In children, convulsions and coma are not rare.

As the *sweating stage* approaches, a moisture appears on the forehead, and soon covers the entire surface of the body. Restlessness and discomfort rapidly abate. The temperature falls speedily, pain and headache subside, a free perspiration bathes the surface, the urine flows copiously, thirst disappears, and the patient seems as well as common. An interval now occurs, corresponding to the type of the fever, after which another paroxysm is ushered in with a chill, and a repetition of the symptoms of the three stages is again gone through.

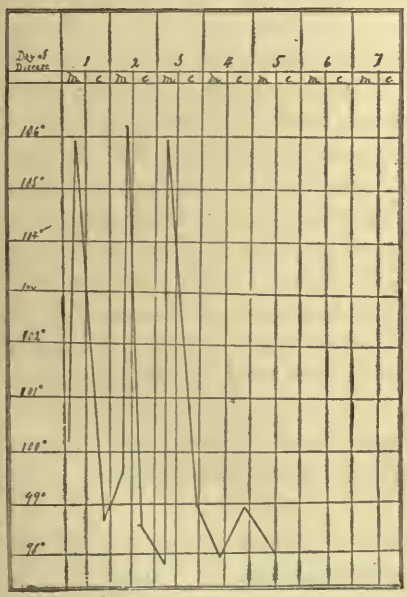


TERTIAN INTERMITTENT.

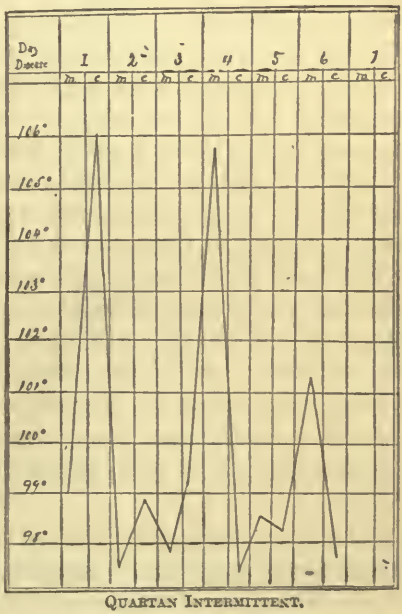
Badly treated, and neglected cases of this disease continue to present a recurrence of the paroxysms, and, after a time, the regularity of the disease may become disturbed, the attacks occurring erratically, the general health of the subject becoming much deranged, the stomach becoming foul, the tongue coated with a pasty white coating, the liver and spleen becoming enlarged, and a complication of jaundice and anæmia being strikingly manifested. Such cases constitute chronic intermittents (malarial cachexia), and demand careful and discriminating treatment, even for the arrest of the paroxysms, as ordinary remedies are quite likely to fail in controlling them.

Diagnosis.—The diagnosis of intermittent fever is very simple. We distinguish it from remittent fever by the fact that there is a complete intermission between the paroxysms, the following attack being ushered in with a chill, while there is not a complete intermission in remittent fever, there being but the initiatory chill, usually, and only a near approach to normal temperature between the exacerbations of fever which follow. There is hardly a chance that intermittent fever will be confounded with pyæmia, as in this disease the chills occur irregularly, and the history of the cases will disclose a different class of etiological factors, in each instance.

Treatment.—That class of practitioners who regard quinine and arsenic as specifics for ague, are usually unsuccessful in its management. Whittaker asserts that the hematozoa disappear from the blood after the administration of *quinia*, and assumes that the specific for the disease is this drug. Depend upon it, they who follow such teaching will leave but sorry results behind, and their patients will finally be obliged to remove from the country or employ other physicians. If they arrest the paroxysms of an ague, which they will, in many cases, there will usually be relapse after relapse, until malarial cachexia will finally be established. Quinia hardly ever cures ague, though it is our best antiperiodic—the best agent to interrupt the paroxysms. *Arsenic* is more permanent in its action, but it should not be depended upon alone to perfect a cure in as stubborn a disease as intermittent fever.



QUOTIDIAN INTERMITTENT.



QUARTAN INTERMITTENT.

Probably it will be best to consider the treatment of the stages separately. The management of the *cold stage* may do much to determine the severity of the remaining portion of the paroxysm. If a patient be placed in an alcoholic vapor bath or a steam bath at the beginning of the chill, the paroxysm may often be almost entirely aborted, the chill quickly passing off, and the hot stage being completely averted. The use of a hot pack, the patient being wrapped in a blanket wrung out of hot water, hot irons or hot ears of corn being placed around him to assist in maintaining the warmth, if nec-

essary, will answer the same purpose, as will any other external appliance which will determine a rapid flow of blood to the surface. In this way, the severe congestion of the internal organs, sure to attend the ordinary course of the disease, is prevented, and the reaction necessarily following, is averted. Those who desire to succeed in such cases can instruct the nurse in the administration of this treatment, and when the physician arrives, his task will be simplified. When the physician is at hand, he may find considerable satisfaction in the use of *nitrite of amyl*, by inhalation, in this stage, from three to five drops being thus administered.

During the *hot stage*, the administration of the *special sedatives*, properly selected and adapted, is to be commended. *Gelsemium* is an excellent agent, as it controls the determination to the brain so commonly attending, and lessens the height of the fever, by promoting secretion from the skin, lungs, and kidneys. *Jaborandi* is also excellent, though it should not be administered in too large doses. Two or three drops of gelsemium or ten of jaborandi may be administered every hour during this stage, in ordinary cases. But gastric irritation may be present, and neither of these remedies will then be appropriate. Here a combination of *aconite* and *rhus tox.* will be demanded, as the sedative action is admirable, while the gastric irritability is nicely controlled by this prescription. Add fifteen or twenty drops of rhus and five or ten of aconite to four ounces of water, and give a teaspoonful every half-hour. Such measures will shorten the length of the hot stage, thus hastening the advent of the stage of decline, and will, moreover, prepare the way for the ready appropriation of the antiperiodic, which it will be necessary to administer during the intermission.

The *sweating stage* brings its own relief, and demands no especial treatment, though the patient should guard against chilling, while relaxed.

An important measure now is to interrupt the periodicity of the disease and prevent, if possible, a recurrence of the paroxysm. If the hot stage has lasted eight or ten hours, we are pretty sure that there will be a chill on the following day at about the same hour as on that of the first attack, and we will prepare to meet it by fortifying the nervous system with a powerful and appropriate stimulant. Our first choice will be the *sulphate of quinia*, though we prefer not to produce too profound cinchonism, since this is liable to leave unpleasant, if not permanent, effects behind, such as tinnitus aurium, deafness, etc. Our sedative has prepared the patient for this remedy, however, so that the small dose will suffice. We will begin seven or eight hours before the time of the expected chill, and administer

three three-grain doses of quinia sulphas, in capsules, one every two hours. This will give us an advantage over the single dose, should the ague prove "anticipating," and should it prove "deferring" the plan will hardly be less effective than the administration of the single dose. If everything is in good condition for the reception of the quinine, we may expect that the chill will not occur. For fear that we may have a tertian, instead of a quotidian, it may now be well to continue the sedative in minute doses for the next day, and repeat the quinine as on the day previous. If, instead of complete success the first day of treatment, we are disappointed in finding the chill to reappear on the second or third, in spite of the antiperiodic, we will repeat it the following time, when success will be almost certain to be the result.

It is good practice to continue the use of the sedative throughout the period of treatment. By this means, the system is prepared for the kindly acceptance and effective action of the antiperiodic, a cure being speedy and certain. In order that quinine may be received kindly by the stomach, and readily absorbed, and its action be unattended by unpleasant nervous symptoms, the skin and tongue should be moist, and the pulse should be open and soft. This condition is to be brought about by the action of properly selected sedatives. *Gelsemium*, being anti-malarial in its properties, as well as sedative, is an ideal sedative in malaria, unless clearly contraindicated by the oppressed, feeble pulse and cold extremities. In this case we will use *belladonna* instead. Use these remedies as follows: ℞ Green plant tincture or specific medicine gelsemium gtt. xx, water ʒiv. M. Dose, a teaspoonful every hour. ℞ Specific medicine belladonna gtt. iii–v, water ʒiv. M. Dose, a teaspoonful every hour. *Aconite* combines well with both remedies, and assists their action. Ten drops of Lloyd's aconite may be added to either prescription, for an adult.

Having arrested the paroxysms, the next measure is to place the system in such condition that there will not be a return of the chills and fever within a few days afterward. In order to fortify the nervous system against probability of this recurrence, I think highly of the third decimal trituration of *arseniate of quinia*. This should constitute a regular medicine for a month, two or three grains being given thrice daily, before meals. The activity of the portal circulation should be looked after, as the congestion resulting here from the ague has most assuredly left a sluggish capillary action, and impaired function. The following prescription will be of service here, to be administered after meals, three times daily: ℞ Polymnia ʒi, chionanthus vir. ʒi. M. Ten to fifteen drops, in water. Keep a watch

on the seventh, fourteenth, twenty-first, and twenty-eighth days. On these days administer a three-grain capsule of quinine with the arseniate of quinia, before each meal. After the twenty-eighth day the disease may be considered cured, provided there has been no paroxysm in the meantime. If there has, there should be a period of complete immunity assured, for four weeks after the last paroxysm.

Masked Intermittents.—Periodical manifestations often occur in malarious districts, which evidently result from malarious influence, and require a similar treatment to that employed in intermittent fever; at least the periodical phase of such cases demands the treatment applied to the periodicity of intermittents. Beyond this we need to apply the special treatment required by the characteristics of each particular case.

Undoubtedly, the etiological factor here is identical with that of intermittent fever, the disease manifesting more of a local predilection.

Supraorbital pain, of intensely painful character, appearing in the forepart of the day and continuing until evening, to pass off and reappear on the following day, at about the same hour, and to recur day after day, is a common manifestation of this kind. Occipital pain is another form of neuralgia which appears periodically, and may be due to malaria. Severe abdominal colic, appearing at some time in the day or night, continuing for several hours and then subsiding, to return the following day or upon the second day, is another form of masked ague. Periodical sciatica, intercostal or frontal pain, tic douloureux, periodical attacks of croup, asthma, diarrhœa, dysentery, hematuria, torticollis, etc., have been observed as periodical manifestations of malarious infection, and relieved mainly by the employment of antiperiodics.

Many times malarious influence complicates other diseases and renders them stubborn to treatment, until the malarial element has been recognized and met. This assertion applies to almost every form of acute disease, and it should be suspected wherever marked periodicity is manifested persistently. Here an antiperiodic should be exhibited at an early period in the treatment.

Periodical muscular pain will demand quinine and *cimicifuga*. Periodical tic douloureux will demand quinine and *piper methysticum*. Periodical pain in the middle ear will demand quinine and *pulsatilla*. Periodical dysentery will demand quinine and *ipecac*. Periodical croup will demand quinine and *aconite*, etc. In each case, the antiperiodic should anticipate the paroxysmal attack in the same manner as in the treatment of that of intermittent fever. In case a chronic condition of the kind becomes established, the treatment applicable to chronic intermittents will be applicable here.

Remittent Fever.

Synonyms.—Bilious Remittent Fever; Jungle Fever.

Definition.—A form of malarial fever in which the temperature remits, but does not intermit, and the exacerbations are diurnal in character, invariably.

Etiology.—This form of malarial fever is most liable to occur in marshy districts, the malarial poisoning being intense in quality, or else the patient manifesting a marked susceptibility. It is undoubtedly due to the same character of poison as the infection of intermittent fever, one form frequently merging into the other, when neglected or badly treated. Remittent fever is believed to be a manifestation of a more severe grade of malarial infection than intermittent fever, and in severe cases it is not uncommon for a remittent fever to become an intermittent, during convalescence. The severity of this disease is determined largely by climate, that which occurs in temperate regions being mild and tractable when compared with that which occurs in the tropics.

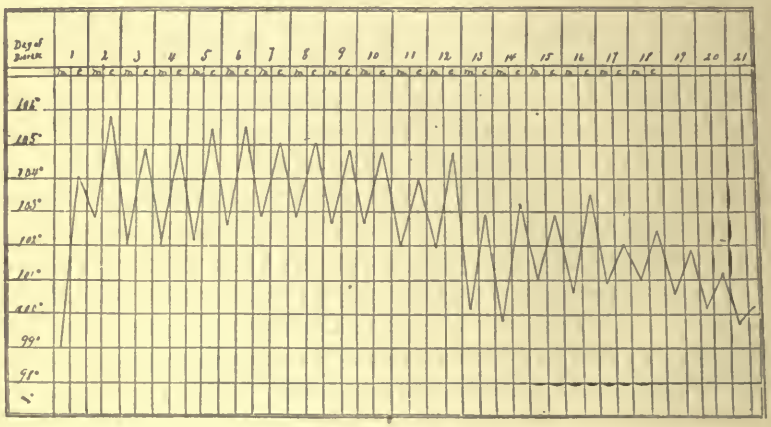
Pathology.—The pathology of remittent fever is almost identical with that of intermittent, the difference being that of degree instead of kind. The cause being the same, we could hardly expect much difference, though the more continuous febrile action would naturally result in greater tendency to pathological changes. Diminution of red globules and loss of fibrin in the blood is common to both forms. Free pigment-granules, however, are more abundant in remittent fever than in intermittent. They are seldom present in intermittent, except in the pernicious forms, while they are almost constantly present in all forms of remittent. This pigment is due to particles of hemaglobin, liberated from the corpuscles and floating free in the plasma, it being transformed into granular or crystalline hematoidin.

The *spleen* is enlarged, but not so markedly as in intermittents, suggesting that the splenic congestion may be more the result of the cold than of the hot stage. Pigmentation is here a marked feature of the pathological condition, and the congested tissues are dark and friable in advanced stages of severe cases. The *liver* presents a peculiar appearance, though there is not remarkable congestion; the peculiarity is the color, the organ presenting a *bronzed* hue, throughout its substance, the "bronzed liver" being regarded as characteristic of this disease. However, it is occasionally, though rarely, met with in intermittent and pernicious fever.

Changes occur in the mucous membrane of the alimentary canal. The mucous membrane of the stomach and small intestines is congested and softened, and the glandular structures are enlarged.

There may be ulceration in places, though not of the character of that of typhoid fever.

Symptoms.—There is usually an intimation of the approach of this disease manifested by a sense of oppression in the epigastrium, with headache and general malaisé, for two or three days before the chill occurs. The tongue often accumulates a pasty-white coating, and the appetite disappears, during this time. Though marked, the *chill* is not as protracted as that of intermittent fever, and there is not the tremulousness and shaking, chattering of the teeth, etc., which occurs in ague; and the sensation of coldness is general in its inception, not coming on by creeping along the spine in the beginning, as in intermittents. Symptoms of nausea may be manifest even during the chill, but if not, they are apt to appear soon after the hot stage begins. Thirst is almost always an urgent symptom.



REMITTENT FEVER.

The *temperature* may advance two or three degrees during the chill, but it rises rapidly after the hot stage begins, and within ten or twelve hours after its inception the thermometer may mark 105° or 106° F. The skin becomes dry and hot, the pulse increases in frequency, ranging from 100 to 120 per minute, being full and bounding in individuals of fair reactive power. The face is flushed, the eyes suffused, the conjunctiva congested, and the patient is restless and uneasy. Muscular pain is now a common symptom, there being aching in the back and limbs, in many cases, or severe hemicrania; in many cases there is full, throbbing headache. As the headache continues, the gastric symptoms are apt to become aggravated, the nausea and vomiting being one of the most unpleasant features of the case, though this symptom is not invariably present. Epigastric pain is a very distressing symptom, in many of the cases in which

vomiting is present, and this is aggravated instead of relieved by vomiting. The material ejected by vomiting becomes greenish as soon as the contents of the stomach have been evacuated, this "bilious" material having given the name "bilious fever" to the disease, in many quarters. Constipation of the bowels is a common symptom; though, if it be not arrested by treatment, diarrhœa sets in later.

After ten or twelve hours, a slight perspiration appears on the forehead and gradually extends over the body, while many of the unpleasant symptoms disappear. The gastric symptoms now become ameliorated, the restlessness and headache subside, the temperature falls several degrees, and the patient experiences a number of hours of comparative comfort—surcease from the sufferings of the hot stage. But the hot stage does not entirely disappear. The thermometer will indicate a temperature above normal during the most comfortable period, and, on the following day, the fever will return, and all the discomforts and suffering of the preceding day will be repeated, with aggravation of many of the most important ones, unless proper treatment has been promptly begun. The increase of fever, with the attending symptoms, is termed the *exacerbation;* the decline of the fever and attendant symptoms, the *remission.*

It will be noticed, however, that the decline of the fever—the interval between the exacerbations—is not followed by a chill before the following rise of temperature occurs. There is a regular rising and falling of the fever, day after day, with but the one chill—that which initiated the attack. This fact will distinguish the disease from ague.

Not all cases of this fever are so severe as the symptoms here given might indicate. Often the symptoms are much more mild, and the disease occurs without the manifestation of severe gastric disturbance. Again, proper treatment, begun early, will often mask the full development of the case. Sometimes the febrile symptoms are not marked, the temperature of the exacerbation not reaching more than 103°, the corresponding symptoms being much modified. Here, however, there will likely be considerable nervous prostration, with gastric irritability, attended by nausea, insomnia, anorexia, irritable stomach, indicated by pointed tongue, with reddened tip and edges, small, wiry pulse, and idiosyncrasy against the kindly reception of quinine as an antiperiodic. Such patients are usually delicately organized, and require an entirely different course of treatment from that which is applicable to the more sthenic cases.

Diagnosis.—The diagnosis of malarial remittent fever is not difficult, though it would be if the temperature curve were to be

depended upon alone—unless a microscopical examination of the blood were made to detect the plasmodium malariæ. The remissions are not only shown by the thermometer, but there is a clinical picture of relief afforded during the remission, not observable in remittents of non-malarious origin. After the use of sedatives for a short time, the reception of quinine is kindly and its action effective, while this is not the case in other remittents. The vomiting and bilious symptoms are present in yellow fever, but here there is but the one paroxysm, and its contagious character is soon developed. The single chill in the beginning will differentiate it from intermittent fever, which is characterized by a succession of rigors.

Prognosis.—Simple remittent fever, of malarial origin, is not a grave disease, under proper treatment. It hardly ever proves fatal, though often prolonged and aggravated by improper management. Ordinary cases ought to be convalescent within four or six days.

Treatment.—Simple cases of remittent fever, in temperate regions, are usually readily arrested within the first week. *Quinine* is the remedy which arrests the paroxysms, it only being necessary to prepare the patient for its ready appropriation. With secretion arrested, absorption by the stomach is impaired, and a stimulant to the nervous centers, like quinia, when absorbed, acts as an irritant, instead of promoting normal functional action. The administration of the antiperiodic, then, without first preparing the patient for it, produces unpleasant effects, and does not yield as good results as when the way is properly paved for its use. A properly selected *sedative*, then, is the first desideratum, and this should be administered in small doses, frequently repeated, throughout the course of the disease. It is hardly necessary to enter into details as to the proper sedative to be selected. *Gelsemium* will be recollected as the ideal sedative where there is the full, bounding pulse, with bright eyes, contracted pupils, flushed face, etc. *Jaborandi* will, however, often afford better satisfaction in these cases. The specific medicine will hardly fail to accomplish good work here.

The gastric complication which attends many cases will, however, modify this selection, the condition demanding a remedy which will serve the double purpose of a general, as well as gastric, sedative. *Aconite* is here the leading remedy as a sedative, while *rhus tox.* combines well with it, as it is one of the most reliable gastric sedatives we possess, the same property belonging, in not a little degree, to aconite.

The size of the dose to be employed in these cases is an important consideration. Too much of a sedative action would be likely to embarrass instead of assisting the cure, and too little would only

be temporizing. The ability of the stomach to tolerate the medicine should also be considered. Two drachms of specific jaborandi (or some other reliable preparation) should be added to four ounces of water, and a teaspoonful ordered every hour. Where gelsemium is employed, the dose may vary from one-half drop to two drops, repeated every hour. In using the aconite and rhus combined, from five to ten drops of aconite and fifteen or twenty drops of rhus in four ounces of water, will meet the requirements, the dose being a teaspoonful every hour, as with the other remedies named.

Muscular pains should not be neglected, as they are common complications (or conditions) of this disease. Often we may be able to combine *cimicifuga* or *rhamnus californica* with the sedative mixture, and arrest this at an early period of treatment. Where it is stubborn, a decoction of rhamnus should be given separately, in full doses, until a laxative effect is produced; or, in the absence of the bark, a good fluid preparation may be administered in fifteen- or twenty-drop doses, until the same object is attained. In many cases I have been well pleased with the action of a combination of *phenacetin* and *arseniate of quinia*. I employ it in capsules, each containing three grains of phenacetin and two of arseniate of quinia, the capsule being administered every three hours, until the object,—the relief of the myalgia,—has been accomplished. However, the remedies for pain should not interfere with the steady use of the appropriate sedative.

During the remission is the proper time for the administration of the *antiperiodic*. This will ordinarily be *quinine*, and it will usually act promptly and effectively, as well as kindly, in interrupting the exacerbations and subduing the disease, when the tongue is moist and cleaning, and the pulse full and soft. We need not wait for decided evidence of this condition after the sedative has been administered for twenty-four hours, for this will almost certainly insure the kindly appropriation of the quinine. Three grains of this remedy, in capsule, may be administered as soon as the remission becomes well marked, and repeated every hour until three doses have been taken, or until a marked rise in temperature is manifested. The exacerbation now being again initiated, the antiperiodic should be withdrawn until the next remission, when it should be repeated as before. While the antiperiodic is being administered, the sedative may be continued, as well as during the exacerbations, throughout the course of the disease.

There are certain *septic* conditions which may interfere with the kindly appropriation of the antiperiodic, at least with its curative effect, and these should not be forgotten, as they are rather common

in remittent fever. The most important condition of this character, as it is the common one, is that marked by the pasty white coating on the tongue, suggesting excessive acidity of the gastro-intestinal canal. Such a condition will demand the administration of *sulphite of sodium*, which may be given in capsules, from half a grain to a grain at a time, repeated every three hours, until the tongue has begun to clean. This must be a cardinal feature of the treatment when the indication for it is well marked, if success is to be expected. The proper action of all other medicine will depend upon this measure. Sometimes an *emetic* will be demanded as an initiatory measure of the treatment. Here the tongue will be heavily loaded at the base, there will be gaseous eructations, and other evidences of chylopoietic torpor. In order to insure proper results here, the patient should have a thorough emetic, and it may be necessary to repeat it on the following day. A yellow-coated tongue suggests a cathartic.

The treatment of the *nervous* form of remittent fever requires the use of *aconite* and *rhus tox.* as a sedative. The gastric irritability, indicated by the pointed tongue, with reddened tip and edges, demands this treatment, and the nervous irritability, restlessness, nocturnal delirium (which occasionally attends), and small, rapid pulse, all suggest this sedative. As the remissions are not marked, and as quinine does not seem to act well in interrupting the fever, it is well to avoid it as a remedy here entirely, and depend upon *arseniate of quinia*, 3x trituration, as this is acceptable, easily tolerated, and very effective, though not as rapid in its influence as quinine, in appropriate cases. The sedative being administered in the doses already suggested, three grains of arseniate of quinia may be administered every four hours, until the disease is arrested. Sometimes a typhoid condition seems to approach; the patient becomes somnolent, semi-delirious, and prostrated, and *echinacea* may prove the best sedative. The combination of *alstonia* with the arseniate of quinia is often a good measure, the combination being that of two or three grains of alstonia with the same quantity of arseniate of quinia 3x, in capsule.

During the height of the exacerbation, sponging of the surface, at occasional intervals, will be advisable, and cold cloths on the forehead, or sponging the head and fanning, will assist in relieving the unpleasant head-symptoms. The *diet* should be light, liquid food being preferable, and it is needless to urge much upon the patient, as the probability of an early termination of the disease will render it important that the stomach should not be burdened with food during the height of the disease. Rice-water, milk, toast, and other light articles, may be taken during the remissions.

Pernicious Malarial Fever.

Synonyms.—Congestive Fever; Tropical Typhoid Fever; Pernicious Fever.

Definition.—A malarial disease, characterized by severe anatomical lesions, and attended by rapid prostration and death, unless promptly treated during its early stages. A malignant form of malarial disease.

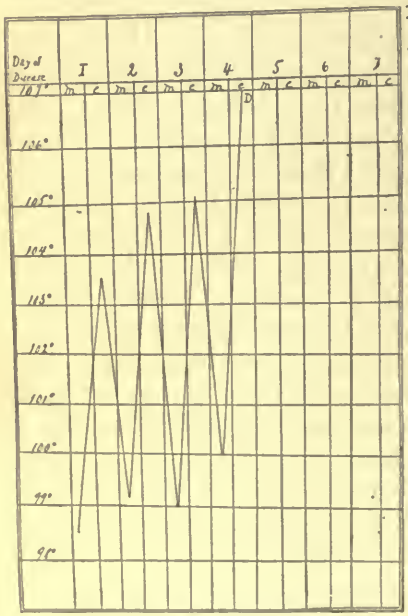
Etiology.—The cause of this disease is identical with that of other forms of malarial fever, the malignant character being due to a predisposing condition of the system, to an average high temperature, or to epidemic influences. It prevails quite extensively where the average temperature reaches for a time 65°, but it may occasionally be observed in more temperate regions, though not with such marked fatality. I have seen several well marked cases of the comatose variety of this disease in Ohio, and one case of the delirious form in Missouri, though all were promptly amenable to treatment but one, a comatose case, in which treatment was rejected early.

Pathology.—The morbid lesions of pernicious malarial fever are less marked in autopsies than those of malarial cachexia. In the *blood* the abundant destruction of red corpuscles is notable, as well as the large amount of black pigment derived from them, found particularly in the visceral capillaries. A crenated condition of the red corpuscles may be found upon microscopical examination, and the relative number of white corpuscles is large, this being due, not to any marked increase in number, but to the rapid destruction of the red corpuscles which has occurred. The *spleen*-changes, common to acute malarial affections, are also present. The organ is swollen, soft, and almost black in color, the fluid contained being dark and watery. The *kidneys* are hyperæmic and pigmented, and the *heart* is pale and flabby. The *lungs* are congested, the lower lobes being especially engorged. The *nervous system* is more or less involved, the brain being hyperænic, the ventricles filled with serum, and the cerebral capillaries blocked with débris of broken-down corpuscles, hematozoa, and pigment.

Symptoms.—Pernicious fever may assume a variety of types, and it may appear in the beginning as an intermittent or remittent form of fever. It may begin as an ordinary intermittent and assume the character of pernicious fever after one or two paroxysms; or it may begin as an ordinary remittent and continue so for several days before the pernicious symptoms are manifested. In other cases, a premonitory chill initiates the symptoms, which immediately appear as those of one of the varieties of pernicious fever.

Several varieties of this fever have been described, the disease seeming to manifest itself in a different manner in different localities, or in different seasons. It has been asserted that epidemic influences operate at different times to determine the prevalence of particular varieties. Though the pathological lesions are but slightly varied in different cases, the symptoms are of a marked diversity of character. Seven prominent varieties may be mentioned, viz., the comatose, the delirious, the gastro-enteric, the icteric, the algid, the hemorrhagic, and the colliquative.

The symptoms of the *comatose* variety are marked almost from the beginning. An ordinary attack of intermittent or remittent fever may ensue, but the hot stage is attended by a *comatose* condition, from which it is almost impossible to arouse the patient. He lies in a state of stupor and unconsciousness, upon his back, his face upturned, flushed, eyes congested, pupils dilated, pulse slow and labored, respiration slow, deep and stertorous. The *temperature* now ranges from 105° to 107° F., in the axilla. There is loss of power of the sphincters, involuntary evacuation from the bladder occurring, or, instead, the urine may be retained, and the bowels move involuntarily. The patient passes deeper and deeper under this influence, the comatose state becoming more marked for ten or twelve hours, when a moisture may appear upon the forehead, a perspiration break out over the entire surface, and the patient awake, perspiring profusely. An intermission or remission now follows, when a more severe attack is attended by all the symptoms of the former comatose condition, in an aggravated form, and the patient may pass into a fatal stupor. Or, he may pass into a condition of apparent death, remain in that state for hours. But almost invariably fatal results follow, the prognosis becoming more unfavorable with each succeeding exacerbation.



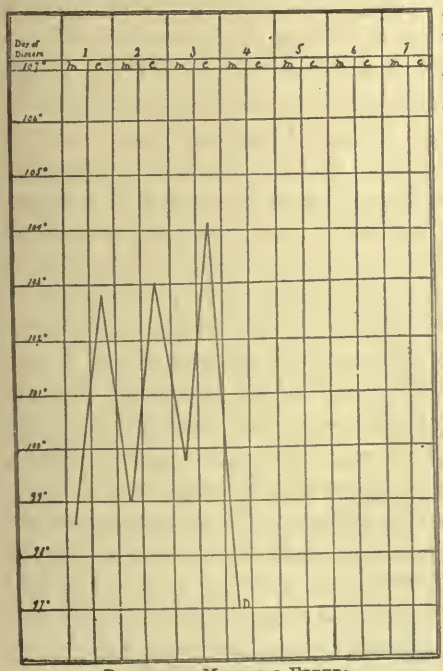
PERNICIOUS MALARIAL FEVER: COMATOSE VARIETY.

In the *delirious* form, after the patient passes into the hot stage, active delirium sets in, the patient raving and tossing, and finally, as the delirious stage advances, attempting to get out of bed, and resisting opposition by struggling furiously. This may last for hours, the

patient screaming wildly and endeavoring to escape from the room. The face is flushed, the eyes congested, the pulse full and bounding. The temperature may be very high, often reaching 107° or 108° F., in the axilla. This may go on for hours, the hot stage finally terminating in a short remission, or the patient sinking into a condition of fatal coma. If a remission occurs, another stage of delirium soon follows, and a fatal termination is almost certain to result.

In the *gastro-enteric* variety, the prominent feature of the hot stage is violent vomiting and purging. The efforts are attended by great prostration, by cramps in the extremities, weight and burning in the stomach, and intense thirst. The symptoms are similar to those of Asiatic cholera, except that the evacuations are not the rice-water discharges of that disease, they usually being blood-stained in appearance, or like the washings of raw meat. Sometimes, however, the evacuations resemble those of cholera. Authors describe a peculiar respiration attending this disease, it being a double sighing inspiration, followed by a double sighing expiration. Collapse and death are likely to follow the first attack.

The *algid* variety is what is commonly known as "congestive chill." The surface of the body becomes of marble coldness after the initiatory chill is past and the hot stage of an intermittent or the exacerbation of a remittent has begun. The surface of the body begins to grow cold, and finally only the surface of the abdomen is warm to the touch, while the patient complains of gastric heat and thirst. The rectal *temperature* may run as high as 104° or 105° F., or in some cases, even higher. The skin becomes clammy, the pulse slow and faltering, the axillary temperature falls as low as 88° or lower, the extremities are like marble in coldness, the tongue is cold, white, and clammy, the breath is chilly, and the voice becomes feeble and indistinct. Sometimes there is coma in this form, and sometimes delirium. Usually the patient is conscious, however, and does not realize much



PERNICIOUS MALARIAL FEVER: ALGID VARIETY.

discomfort, except that of exhaustion and great internal heat. The common termination of this condition is death, the disease going rapidly forward to a fatal termination.

The remarkable symptom in the *icteric* variety is the yellow tinge of the skin, marking a profoundly jaundiced condition. The disease begins with a severe chill, which is protracted, and during which a remarkable yellowness of the skin develops. This jaundice involves the conjunctiva and entire skin, and gradually deepens, until the surface is markedly greenish-yellow. Vomiting usually sets in at an early period, and this is attended by a bilious diarrhœa. Severe headache, pain and fullness in the spleen and over the kidneys, numbness in the limbs, and great restlessness, attend. The urine is scanty and dark-colored, so much so as to constitute the condition termed "melanuria." The pulse is small and frequent, and respiration labored. As the hot stage arrives, the pulse becomes full, rapid, and bounding, the skin very dry and hot, the temperature high (106° or 107°), the headache bursting, and the thirst intense. Within three or four hours, this stage is liable to terminate fatally. If the skin becomes moist, a sweating stage comes on, and a remission becomes established. But each succeeding paroxysm becomes more and more severe, unless the disease is arrested by treatment, until a fatal termination ensues. This form occurs endemically in certain localities, prevailing whenever any form of pernicious fever appears there.

The *hemorrhagic* variety is characterized by *hemorrhage* from the *kidneys*. The fever may be intermittent, remittent, or continuous in character. The subjects are usually victims of profound mercurialism, of chronic malarial cachexia, or chronic alcoholism. It occurs in tropical regions, and is attended by symptoms of jaundice, it being a combination of the icteric and hemorrhagic states. It is common in the swampy regions of the Southern States, Alabama, Mississippi, Arkansas, and Louisiana being the theater of its action, where low marshy regions along the rivers furnish the requisite conditions for its development. Hemorrhage from the kidneys may occur in almost any region and attend malarial disease, but in many cases it is not of the malignant character that is comprehended by the hematuria of pernicious hemorrhagic fever. This disease resembles yellow fever in many of its characteristics, but it does not occur epidemically, and there is more splenic enlargement.

Blood appears in the urine sometimes during the cold stage, but more profuse hemorrhage is apt to occur during the period of febrile action. The urine is dark, acid, and albuminous, and contains tube-casts and blood-corpuscles, there being a copious sediment deposited upon standing. Where a marked remission occurs, the hemorrhage

may cease for the time being, but it returns upon the onset of the following paroxysm or exacerbation. Icterus, vomiting, severe headache, and pain in the back and loins, attend. The respiration is sighing and oppressed, the decubitus is dorsal, the tongue is first broad and moist, and covered with a pasty white coating, but later, after icteric symptoms have developed, the coating becomes yellow. In fatal cases, it turns dry and brown, or becomes covered with black sordes. The pulse is full and oppressed. The bowels are often constipated in the beginning, but a watery diarrhœa appears later, the dejections being yellow, green, or black. The temperature runs from 106° to 108° during the hot stage, and this continues for several days, the decline being attended by a period of profound adynamia, which lasts for several days. This disease is very apt to terminate fatally.

In the *colliquative* variety, a prostrating *sweat* follows the hot stage, and continues during the intermission. The pulse is feeble and oppressed, the respiration is sighing and labored, and the succeeding exacerbation begins with the patient very much prostrated and exhausted. After two or three such periods, he sinks and dies of exhaustion. In other cases, severe hematemesis or hematuria may occur during the sweating stage, and rapid prostration follow, often with fatal results.

Diagnosis.—There is little probability of confounding this disease with anything but yellow fever, and this might be the case in the icteric form, if it were not recollected that yellow fever occurs epidemically, and that it is contagious, which is not the case with icteric pernicious fever. The gradual rise in temperature and insidious invasion of the acme stage, with the abdominal symptoms, would usually distinguish typhoid fever. When gastro-intestinal symptoms are present in pernicious fever, they are violent in character, resembling those of cholera or yellow fever. The absence of an epidemic will exclude danger of confounding it with either of these diseases.

Treatment.—The violence of the attack in pernicious fever will usually interfere with the selection of remedies on the lines usually suggested in specific medication. However, prominent indications in this direction should not be disregarded, as some prominently indicated remedy, such as *sodium sulphite*, might be the turning pivot upon which the life of a patient would depend. The simple administration of *quinine*, as advised by some authors, is certainly not all that can be advised, though it is admittedly an important item of the treatment.

The *comatose* variety should be treated by a *hot bath*, which may

be administered by packing the patient with flannels wrung out of hot water, while full doses of *jaborandi* are swallowed until perspiration has been established. If the patient cannot be aroused sufficiently to be induced to swallow the drug, one-fourth grain of *pilocarpin* may be administered hypodermically, and repeated in an hour, if a decided action is not manifested by that time. As soon as the action of the drug is manifest by the indication of perspiration, twenty or thirty grains of *quinine*, either the bisulphate or sulphate, should be administered hypodermically, and the injection should be repeated every two hours until the patient has passed under the influence of the drug, and is out of immediate danger. As soon as the attack is arrested, means should be taken to remove such specific conditions as are prominently manifested by the individuality of the case. Portal congestion, a condition almost certain to be present, should be met with *polymnia* or *carduus marianus;* gastric conditions should be corrected, and, as there will be likely to be acidity with sepsis, *sulphite of sodium* will meet the indication, "tongue loaded with pasty-white coating." Other specific indications should be met, as far as possible. Gastric irritation may be treated with *aconite* and *rhus tox.* If practicable, the patient should be removed from the intensely malarious district into the most salubrious neighborhood possible, until time has been allowed for recuperation.

In the treatment of the *delirious* variety the *hot pack* is especially applicable, and here also the hypodermic use of *pilocarpin* will be the most prompt and effectual method of bringing the circulation under the influence of a sedative and equalizer, and preparing the way for the appropriation of *quinine.* Whatever form or variety of pernicious fever we may encounter, there is an overpowering accumulation of the provoking element in the circulation, which oppresses the vasomotor centers, and causes congestion of internal organs. To equalize the circulation then is the first requirement, and nothing will do this so readily and promptly, considering the facility of administration, as pilocarpin. A flannel blanket wrung out of hot water and wrapped around the patient as hot as can be borne without scalding, answers the double purpose of confining a struggling patient, and of equalizing the circulation and lowering the temperature. Under the influence of these measures, the patient will soon become calm, and fall into a sleep, from which he will awake apparently almost recovered. However, the antiperiodic must be promptly administered, as a second paroxysm is liable to appear, in worse form than the first.

In the *icteric* form, the addition of *polymnia uvedalia* is an important aid to treatment, and here the dose must be large, fifteen or

twenty drops of the specific medicine every hour or half hour not being too much. There is no objection to combining *chionanthus* with it, though the action of this drug is too slow for the speedy effect here desired.

There is no objection to the employment of the same treatment in the *colliquative* form of this disease, for, though there may be profuse sweating, there is want of proper circulation—an oppression of the nervous centers which regulate the circulatory system—and *pilocarpin* will afford good results here, establishing more of a salutary condition in the sudoriparous glands, and preparing the way for the kindly action of the antiperiodic.

It must be recollected that many cases of this kind are attended by persistent vomiting, of a character which is very difficult to speedily control; therefore, hypodermic medication offers the most rational means for the administration of remedies, as we are assured that it will be retained until its effects have been produced. Any one of the special sedatives may be employed hypodermically, aconite, belladonna, veratrum, or gelsemium acting as effectively that way as per mouth, though aconite and belladonna, if not veratrum, better be omitted, since the large dose of such powerful remedies is never advisable, and the large dose must be administered here in order to get speedy results.

It will be observed that little discrimination is made as to the treatment of different varieties, and such is not necessary. For, though the symptoms may vary widely, there is such a sameness in etiological and pathological respects that it would be folly to waste valuable and often vital moments treating accidental symptoms. These may all be hunted up and prescribed for after the patient's life has been saved from immediate jeopardy, and we have a case of malarial cachexia to treat.

Hypodermic Injection of Quinine in Malarial Fever.—On this subject I will quote an article by H. Martyn Scudder, M. D., published in the *Medical Record* in 1885:

"About a year after my arrival in India I was placed in charge of a general hospital and dispensary, situated about seven miles from Madras, in a town which, with its suburbs, contained a population of over thirty thousand inhabitants. In the year 1876 I was induced, by several articles published in the British and Indian medical journals, to try extensively the treatment of intermittent and remittent fevers by the hypodermic injection of sulphate of quinine. As over nine thousand patients were annually treated in this hospital and dispensary, I was enabled to try this treatment on a large scale.

"At first I employed a solution made with ordinary sulphate of quinine and dilute hydrochloric acid, and I was astonished at the wonderful result produced by these hypodermic injections. Cases that had been taking twenty and thirty grains per diem by the mouth without any apparent effect, were cured at once by the injection of from eight to twelve grains. In the first 100 cases, subjected to this treatment, I had 5 cases of abscess following the injections. Not being satisfied with this result, I determined to try a solution of the quiniæ sulph. solubil. (an English preparation very similar to our bisulphate), prepared with a little tartaric acid. I found this a great success—I might almost say a perfect success. I have used this injection in over two thousand cases without any bad effects, with the exception of one case of small abscess. Even in this case I am inclined to think there would have been no abscess if the patient's arm had been firmly held, and if the operator had not been interrupted by the violent movements of the chill.

"During the last three years of my residence in India I was settled on the Neilgherry Hills, where I had a good opportunity of carrying on this mode of treatment by hypodermic injection, and of ascertaining that it was as efficacious with Englishmen as it had proved to be with the natives. Near the tops of these Neilgherry Hills, at an elevation of about six thousand and eleven thousand feet above the level of the sea, are situated the two large sanitaria of South India, with an English population of from five thousand to ten thousand, according to the season of the year. The climate of these hills at this elevation being cool and healthy, Europeans are able to live there in comfort and bring up their families. A great number of English gentlemen are residents upon these hills, being engaged in the cultivation of coffee, tea, cinchona, or 'planting,' as it is termed. To work their large and valuable estates great numbers of native laborers are employed. These plantations are situated on the slopes of the hills, at an average elevation of three thousand five hundred feet, and therefore below what is called "fever range" that is, the hilly or mountain tracts of South India are generally infested with malaria until you reach an elevation of over five thousand feet. At certain times of the year it is unsafe to sleep even for a single night on these estates. The native laborers are obliged, however, to live down there, and are therefore constantly prostrated with fever. I have frequently been called to visit one of these estates, and in a single morning would often have to administer hypodermic injections of quinine to over fifty of these native laborers. In addition to constantly and regularly employing hypodermic injections of quinine in the treatment of malarial fevers, I

administered quinine in this way in puerperal septicæmia, where I found it had a very beneficial effect.

"A considerable number of cases of puerperal septicæmia occurred in the lying-in ward of the General Hospital and Dispensary already alluded to. The native doctors and midwives have no real knowledge of anatomy and physiology, and they often employ most violent means to hasten difficult labors. It is therefore not at all surprising that many cases of septicæmia and peritonitis result.

"I have already mentioned that the solution generally used was composed of quiniæ sulph. solubil., tartaric acid, and distilled water, the strength of the solution varying from fourteen per cent, to twenty per cent. With adults I usually injected two syringefuls (that is, from five to eight decigrammes) into the upper and outer part of the arm, or in the back of the shoulder, pushing the needle well down into the subcutaneous tissues and even into the muscle. The pain produced was always trifling and of short duration. Sometimes a little redness and very slight swelling occurred, but soon disappeared. I once administered to an English army officer, taken suddenly with a congestive chill, eight syringefuls of a sixteen per cent solution in twelve hours, with the very best of results, except that he was somewhat deaf for a day or two. Of course I do not mean to assert that this treatment by hypodermic injection always drove the malarial poison entirely out of the system, or effected a permanent cure; but one or two injections nearly broke up the fever, and effected a cure at the time, so that a patient would have no return of the fever, unless he exposed himself anew by visiting a locality where malarial fever was rife, or allowed his general health to run down. I had two patients—wealthy English gentlemen—who were accustomed to come every few months and get me to give them a hypodermic injection of quinine as a prophylactic measure when they were about to visit a notoriously feverish locality. During the eighteen months that I have been practicing here in Chicago, I have made the use of hypodermic injections of quinine several times, with perfect success. In conclusion, to show what perfect confidence I have in this mode of treatment, I have only to mention that I have had quinine injected into my own arm on two occasions."

Typho-Malarial Fever.

Synonyms.—Continued Malarial Fever; Remitto-Typhus Fever.

Definition.—A term applied to forms of malarial fever presenting features of a continued type, such as nervous prostration, absence of appreciable relief during remissions, tongue indications

foreign to those of pure malarial fever, and pronounced aggravation from the influence of quinine.

Nature.—The term "typho-malarial fever" has been something of an omnibus, under which quite a variety of conditions have been grouped, the only specific characteristic common to all being the manifestation of malaria as exhibited by marked periodicity in the beginning. Some authors discard the term altogether, on the ground that true typho-malarial fever is a complication of typhoid fever with malaria, and that there is no excuse for classifying a new disease. If this were true, it might be wise to adopt such a course; but everybody who has had much experience in malarious regions knows that cases of fever frequently occur here which are different from pure malarial fever, in their tendency to run a continued course (so far as the unpleasantness of their symptoms is concerned), with typhoid symptoms, in spite of antiperiodics and other treatment which will arrest ordinary malarial attacks, and which are not true enteric fever, as there is nothing in their history to indicate a possibility of such infection, and abdominal symptoms are the exception rather than the rule; and then this is an accidental complication, instead of an important feature of the disease.

Other diseases than typhoid fever may be complicated with malaria, such, for instance, as dysentery and pneumonia, and when these diseases occur as an epidemic where malarious conditions are prevailing, we may have typho-malarial dysentery or typho-malarial pneumonia; but this kind of a complication is not the condition for which the name is here intended.

The name had its origin about the time of the last American war, when the soldiers of the north were stricken on the banks of the Chickahominy with a severe and fatal disease, the symptoms doubtless being due partly to the paludal influence of the surrounding swamps, and partly to such anti-hygienic influences as the deposition of much fecal material upon the surface, to contaminate neighboring springs and other sources of supply of drinking water, with the typhoid fever bacillus. Doubtless this was a combination of malaria and true typhoid fever, a condition which it is not the intention to discuss here. I have appropriated the name for an entirely different disease.

The object of this article is to discuss a malarial fever in which there are typhoid symptoms, without combination of specific typhoid fever conditions—a condition often, but not always, characterized by marked periodicity, occurring in malarious districts, but in which the measures which arrest ordinary malarial fevers prove futile, and in which the patient passes through various stages of septic fever,

as indicated by the tongue-changes and accompanying symptoms, the disease running, in spite of treatment, from fourteen to twenty-one days.

The following notes on the parasite of malaria, by U. S. N. Surgeon Craig, suggest the reason for the disappearance or occasional absence of marked periodicity, as well as throwing light upon the etiology of malarial diseases in general:

"It is during the apyrexial period that the organisms grow, producing few or no symptoms. It is only when the stage of segmentation is approached that the temperature begins to rise, and reaches the acme about the time that segmentation has been completed; then declining to normal, the paroxysm lasting an indefinite period, depending on the potency and quantity of the toxine evolved.

"In the ordinary tertian, and double tertians, the quartans and their combinations, the length of the paroxysm averages from about six to ten hours.

"In the æstivo-autumnal, or the remittents, as they are commonly called, the duration of the paroxysm is much longer, averaging from eighteen to twenty-two hours, or even as long as thirty-six hours in some cases; thus, it is the overlapping of these paroxysms which gives the irregularly continuous fever; that is, the toxin produced by one set of organisms does not become eliminated before the advent of a fresh quantity of toxin by the succeeding set of organisms.

"In the pernicious and congestive forms of these fevers, the toxin is in such a virulent form that sometimes one paroxysm is sufficient to cause the death of the patient.

"It is not the high temperature which kills in all cases, for we find that in some cases the temperature becomes subnormal, 96° F. or less, and remains so, the patient dying in a condition of coma, just as in the hyperpyrexial case."

Etiology.—As will be inferred from the foregoing, the etiology is somewhat obscure. Undoubtedly the plasmodium malariæ figures as one of the exciting causes, but there must be an additional factor, or else it would not be so markedly different from pure malarial fever in its clinical characteristics.

This is evidently not the specific bacillus of true typhoid fever, for the disease cannot be traced to fecal material, nor does it propagate its kind in the manner peculiar to that disease. Indeed, it is doubtful that it is contagious, or even infectious, except so far as the malarial element is concerned.

It is probable that the condition depends upon a peculiar state of the system, brought about by various anti-hygienic causes, such

as depressing influences from mental worry, overwork with anxiety, retained secretions, improper diet, or vitiated air, in combination with the ordinary causes of malaria.

Loomis asserts that sewer gases seem to be the elements which have predisposed to it, in cases which have come under his notice.

Symptoms.—It is difficult to describe the symptoms of this disease, as different cases vary so much. Some peculiarities may be mentioned, however, which are applicable to many of them. They run from fourteen to twenty-one days, in spite of treatment. While an ordinary case of malarial fever, whether intermittent or remittent, can be arrested within the first week, such treatment usually fails to arrest this variety, and it persists in running through its course. Most cases also present marked malarial symptoms in the beginning, in the manifestation of chills and other periodicity, and lāter grow out of this into a more continued type, as the nervous system becomes more and more involved.

While some observers state that intestinal complication is a common condition, my experience has been that there is little disturbance of the bowels, though in a large class of cases gastric irritation is common. In those cases manifesting intestinal irritation, the season of the year and the character of food eaten prior to the attack are liable to play provoking parts.

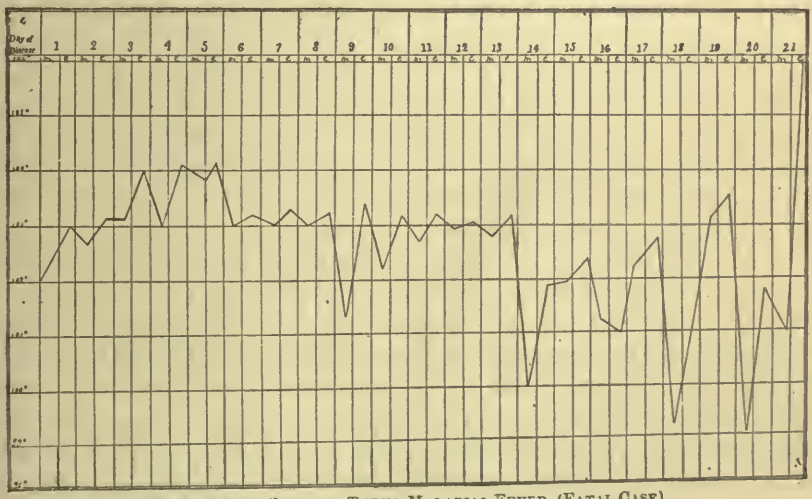
Two prominent classes of cases are found, and I shall divide all cases into these two classes, though occasionally a case will be encountered which cannot be included in either one. We will call them the septic class and the nervous class.

In the *septic* class the attack is usually abrupt and severe. There is a marked and prolonged chill, and this may be repeated every day for three or four days, resembling the paroxysms of an intermittent fever; or, instead, there may be but the one chill, and the remissions and exacerbations of a remittent fever may mark the onset. Instead of the marked relief that attends the intermissions or remissions of an ordinary malarial fever, however, the patient suffers continually, and is not inclined to get out of bed, marked prostration manifesting itself from the start. During the exacerbations, the *temperature* ranges from 103° to 105° F., the pulse is full and strong, hard or bounding, and there is severe muscular pain in various parts of the body, usually involving the lumbar and pericranial muscles; and these pains persist, though not so severely, during the periods of remission.

The *tongue* is coated heavily, with a white or pasty-white coating, the organ is broad and flabby, and there is a putrefactive odor about the breath.

In three or four days, whether antiperiodics have been used or not, it becomes evident that the patient is growing weaker, and that there is no progress toward recovery. He has no desire to get out of bed, though restless and uneasy, and there may be delirium at night. The chills have now passed away, and there is less appearance of a remission in the morning than at first, though the thermometer may indicate fully as much decline of temperature. There are now loathing of food, thirst, severe muscular pain, and marked restlessness. The tongue begins to take on a brownish tinge by the end of the first week, and in two or three days more the coating may flake off, leaving the mucous membrane bare, dark red, and slick—the characteristic beefsteak tongue. Or, the coating may become dry and brown, and gradually wear off by attrition, the beefsteak characteristic not appearing.

Meantime, as the disease progresses, the patient becomes less restless, the night delirium passes off, and a condition of apathy or drowsiness comes on, the patient finally becoming somnolent, and falling into a profound slumber during the morning remissions; and



Temperature Curve in Typho-Malarial Fever (Fatal Case)

soon prolonged sleep comes on, the skin becomes moist, the urine throws down a copious sediment, and convalescence is established between the fourteenth and twenty-first day.

In the *nervous* class, the onset is not so abrupt nor severe. The fever comes on more insidiously, though even here the periodicity of malaria is manifested. The pulse is small and rapid, compressible, or wiry. Prostration is evident early, and is more marked than in the septic class, though the patient does not seem to suffer

severely. The tongue, instead of being large and broad, is narrow and contracted, tremulous on protrusion, and often pointed, and reddened at the tip and edges, in which case there is constant nausea and loathing of food, and sometimes vomiting upon the taking of even fluids, with provoking thirst. The tongue becomes dry and brown early, in many cases, though it may remain moist throughout. A thin white coating may be present along the center of the organ, but it soon becomes clean and slick, or the coating becomes shriveled and brown.

Restlessness is a marked feature of this condition, and the patient is delirious at night, sometimes actively so, and the condition is very much aggravated by quinine or opiates.

The skin is dry and harsh, the secretions generally are arrested, the urine being scanty and high colored, and, when becoming more profuse at the end of the fever, it throws down a copious sediment.

Another form of the nervous variety is the *comatose* form. In this, the subject becomes comatose within a day or two after the attack begins, there being complete prostration of all the voluntary forces. There are mouth-breathing, brown tongue, dilatation of the pupils, involuntary evacuation of urine, and, in some cases, apparent paralysis of the extremities, though motion returns in a few days, when favorable symptoms succeed. The *temperature*, in this form, is not remittent to any marked degree, the curve resembling that of typhus fever. Within a few days, in favorable cases, the remissions become more marked, and the patient rouses from his lethargy, and passes through conditions already described.

Typho-malarial fever, occurring during the heated term, may take on intestinal irritation, and develop diarrhœa or dysentery; or, if bunglingly treated with cathartics in the start, it may develop such symptoms at any time; but when properly managed, the bowels are not usually seriously disturbed, without the operation of some special provoking cause.

Diagnosis.—The absence of the severe abdominal complications of typhoid fever, and the isolated character of the attacks, will exclude this disease from the diagnosis. The delirium is not so severe nor so obstinate to treat as that of typhus, and the former history of the case will usually enable the practitioner to discriminate between them. Cerebro-spinal fever often takes on similar symptoms, but the severe muscular pains of cerebro-spinal fever, the retraction of the head, and the irregular course of the fever, will usually declare the character of that disease. It will hardly ever be confounded with pernicious malarial fever, for the reason that that disease usually occurs in southernlat itudes, while this is a disease of temperate regions.

Prognosis.—There are very few fatalities attending this disease, when proper therapeutic means are employed from the beginning. Though a disease of grave aspect in the early part of its course, rational treatment will almost invariably bring the patient safely through.

Treatment.—One rule should be observed in the treatment of this disease, and that is, to refrain from attempting to break it up, as though it were a case of ordinary malarial fever. Heroic doses of quinine should be left out of the treatment altogether.

In the *septic* variety, where the tongue is heavily loaded at the base, an *emetic*, administered early, may lessen the severity of the conditions which follow, and also prepare the system for the better reception of other remedies.

The broad, flabby tongue will call for one-grain doses of *sulphite of sodium*, administered every two or three hours, until the peculiar coating has begun to disappear. The septic condition should thus be followed with the appropriate remedy indicated by the tongue, throughout the disease. As the tongue cleans and shows the beefsteak color and general appearance, *muriatic* acid will be the proper corrective. If, instead of the beefsteak tongue, the coating becomes brown, with tendency to the deposit of sordes on the teeth and lips, *sulphurous* acid will be more proper.

Where muriatic (hydrochloric) acid is required, we will prescribe as follows: ℞ Dilute muriatic acid ʒi, simple syrup or water ʒiii. M., and order a teaspoonful every three or four hours. Sulphurous acid may be administered in twenty-drop doses, well diluted, every two or three hours, when called for by the brown coating of the tongue.

The proper antiseptic should be accompanied by the proper *sedative*. With the full, strong pulse, we will find *jaborandi* an excellent remedy, though if there is active determination of blood to the brain, as indicated by the bright eyes, contracted pupils, and full, bounding pulse, *gelsemium* may be combined with it or alternated advantageously. ℞ Jaborandi ʒiii, water ʒiv. M., and order a teaspoonful every hour. Or, ℞ Gelsemium gtt. xx, water ʒiv. M., and order a teaspoonful every hour.

The sedative mixture should be administered assiduously until the temperature falls and signs of convalescence appear. Usually, as the disease progresses, the sthenic character of the pulse gives way to a condition of lessened force in the impulse, and the sedative may properly be changed to small doses of *aconite* and *rhus tox.* For example, ℞ Lloyd's aconite gtt. v–x, rhus tox. gtt. x–xv., water ʒiv. M., and order a teaspoonful every hour. Where there is drowsiness, coldness of the extremities, dilated pupils, doughy condition

of the tissues, soft, compressible pulse, and other indications of feeble capillary circulation, ℞ Belladonna (specific medicine or homeopathic tincture) gtt. vi, water ʒiv. M., and order a teaspoonful every hour.

If there be intestinal irritation, with diarrhœa, a better antiseptic than the sulphate of sodium, sulphurous acid, or muriatic acid, will be *echinacea* or *baptisia*. However, if there be the marked tongue indication for any one of these remedies, it should have the preference.

The *nervous* type, bearing evidence of gastric irritation by the elongated tongue, with restlessness, will call for *aconite* and *rhus tox.*, early. ℞ Specific aconite gtt. v, specific rhus tox. gtt. x, water ʒiv. M., and order a teaspoonful every hour. This will constitute the treatment for several days, until all evidence of gastric irritation has passed away. As the beefsteak tongue appearance comes on, the preparation of muriatic acid already described may be made use of, in connection with the sedative mixture of aconite and rhus tox., which must be continued as long as the thermometer indicates the presence of febrile action. Sometimes the tongue will suggest sulphurous acid instead of muriatic, and sometimes echinacea or baptisia will be more effective, the indications for these remedies not being so marked, but the typhoid condition suggesting some agent of antiseptic character.

The treatment already suggested will apply to the comatose variety, belladonna, aconite and rhus tox. being most applicable. Echinacea will be an especially excellent antiseptic here, though where there are prominent indications for others, it should not be used to their exclusion.

Quinine should be omitted until convalescence sets in, and then be administered in small doses, if at all. Better remedies here are *arseniate of quinia* 3x, or ten- or fifteen-drop doses of a reliable fluid preparation of *grindelia squarrosa*.

During the height of the fever, the surface should be sponged with alkaline tepid water each day, as it contributes to the rest and comfort of the patient, as well as assisting the natural efforts to throw off the fever.

The *diet* should be similar to that of typhoid fever, liquid in character, though solid food may be resumed within a much shorter time. Milk, malted milk, gruels, etc., avoiding fruits, may constitute the food for the term of fever, the patient being fed regularly, as a supporting regimen now seems important, to encourage ready recuperation.

Chronic Malarial Fever.

Synonym.—Malarial Cachexia.

Definition.—A chronic malarial manifestation, characterized by anæmia, sallow, waxy pallor of the skin, and splenic enlargement, with attendant indigestion, debility, languor, and other malarial manifestations.

Etiology.—This condition may arise from repeated attacks of acute malarial disease, or it may come on gradually, as the result of long-continued latent malarial poisoning.

Pathology.—The morbid results of this condition differ from those of malarial fever principally in extent. The spleen seems to be the part which suffers the most anatomical change. It is very much enlarged, sometimes filling nearly the entire abdominal cavity, and often being ten or twelve times its normal size, tough, firm, and resistant. The capsule is thickened and uneven, and there may be adhesions to adjacent structures. There is marked pigmentation throughout the entire organ, and hyperplasia or degenerative changes have left their evidences in the structures. The liver and kidneys are similarly altered, though not so prominently so as the spleen. The blood-changes are not so marked as in the various forms of malarial fever, but its impoverished condition is manifested by tendency to dropsical effusions into the cellular tissues and serous cavities. Fibrinous coagula are sometimes found in the arteries, and cavities of the heart. The plasmodium of Laveran, it is asserted, is found in the blood, the crescentric form being the most common.

Symptoms.—Malarial cachexia furnishes us with a great variety of symptoms, but there is such a sameness in the pathological conditions resulting in different cases, that a rational treatment is suggested as applying to the class of cases, and this need not vary so widely in individuals as might at first be supposed. The subjective symptoms are legion. Sometimes they are those of a chronic intermittent, with rather erratic manifestation of the paroxysms, these being attended by unusual symptoms of prominence, such as a neuralgic manifestation,—periodical tic douloureux, sciatica, pleurodynia—and sometimes by extreme gastric or intestinal disturbance, hemicrania, etc. In other cases, the ague type will not be manifest at all, and it may be difficult to detect any evidence of periodicity in the case, unless some acute aggravation arises.

In all cases, there is prominent evidence of disturbance of the assimilative and reconstructive processes. The complexion presents us with a sallow, waxy pallor, characteristic and striking; the patient is debilitated and enervated; palpitation of the heart attends and follows slight exertion; digestion is feeble; the bowels are usually

constipated; the skin is dry and harsh; the tissues flabby and illy nourished; the tongue broad, flabby, and covered with a pasty white coating.

There will be found upon inspection, a fullness of the abdomen over the epigastric and hypochondriac regions, the lungs being crowded upward, and respiration being stuffy and difficult. Sometimes there are decidedly asthmatic symptoms as a result of the pressure from splenic and hepatic engorgement. Hepatic engorgement is so common that icteric symptoms are often more or less manifest, in many cases.

Perversion of the sensibility of the cutaneous nerves is a symptom which is not uncommon. I have seen a few cases where the entire scalp seemed cold to the patient, though not to the touch of the observer, and remained so for months—until restored by electricity and vapor baths. In other cases, there may be tingling and numbness in some portion of the cutaneous surface, notably that upon the outside of the thighs. Whether this is alone the result of malaria or of the abuse of quinine and calomel is not quite clear to my mind, but it usually occurs in those who have been subject to old orthodox allopathic treatment for a considerable length of time.

A certain class of symptoms is always present: more or less vertigo, tinnitus aurium, anorexia, nausea, and difficult digestion. The patient wakes in the morning with a foul metallic taste, dizziness, sense of confusion in the head, and general soreness and stiffness. Myalgic pains, with stiffness of the muscles, are common in this condition, and there is frequently a sense of weariness, constantly present, with nocturnal wakefulness.

Catarrhal symptoms are not uncommon, these manifesting themselves in a catarrhal bronchitis, or in the form of muco-enteritis. Hemorrhages, such as epistaxis, hematuria, menorrhagia, and even hemoptysis, may originate as a symptom of malarial infection. Stubborn menorrhagia, occurring in malarious districts, may sometimes be cured with means directed to the relief of malaria, when apparently more rational measures fail.

Diagnosis.—The existence of malarial surroundings, and the fact that the patient has been exposed to their influence for a long time, connected with the fact that there is anæmia, without any other known cause, will suggest malarial cachexia. Enlargement of the spleen will add testimony to this supposition. Exclusion of serious renal affections will be made by urinary analysis, and careful palpation will serve to exclude hepatic cirrhosis. Periodical manifestations will add to the testimony of malarial origin. Microscopical examination by competent observers will probably detect the

hematozoa of Laveran; however, few practitioners will need to go thus far in order to render a correct diagnosis, even provided they possess the necessary apparatus. Another condition liable to be confounded with malarial cachexia is leukæmia, in which there are enlargement of the spleen and anæmia. Here, however, there is great increase in the proportion of white blood-globules, and treatment for malarial cachexia produces no effect. As leucocythæmia is usually a fatal disease, and treatment for malarial cachexia could not damage the patient, a mistake of this kind would not be serious, at any rate.

Prognosis.—The prognosis will depend largely on the care and attention paid to the management of the case. When possible to remove the patient from malarious surroundings to a higher and more healthy neighborhood for a few months, a much more speedy recovery will probably follow. Advanced stages which have gone on to structural changes in the spleen with amyloid or melanotic degeneration and effusion into the serous cavities, are unpromising. A large majority of the cases, however, may be considered favorable, under the treatment here suggested.

Treatment.—There seem to be rational propositions afforded, by the symptoms and pathology of this disease, for a direct and successful treatment. However, each case of disease will always be an individual one, and no routine treatment need be expected to invariably succeed. But one condition is always present here, viz., *splēnic congestion.* It would almost seem that the cachexia really hinged upon this condition, the congestion being the precursor of the anæmic state. What little knowledge of the functions of the spleen we possess would naturally lend color to this proposition. If the splenic congestion could be averted in the start, it is probable that the cachexia would never result, the other complications following as sequelæ of obstruction to important circulatory channels and necessary blood-making functions.

The proposition resolves itself then into the treatment of a case of chronic (but ordinarily curable) splenic congestion, with attendant incidental complications. Fortunately, the Eclectic materia medica contains a goodly list of remedies which are potent in curing splenic congestion. *Carduus marianus, ceanothus, grindelia squarrosa,* and *polymnia uvedalia,* all possess particular merit in this direction, and, equipped with them, we are prepared to attack the disease at its very foundation. The leading proposition throughout, then, will be to promote normal splenic function, and preserve a free portal circulation by the aid of one or all of these remedies. This accomplished, the remaining part of the task will not be difficult. There

is such a similarity in the action of these remedies that it will not be easy to always discriminate and select the one best adapted to an individual case. However, there are some leading points which it may be well to consider. In many cases the combination of two or more of them may afford better satisfaction than the use of a single one.

Carduus marianus is adapted to the treatment of rather recent cases, in which there is a mental complication bordering on hypochondriasis; the patient is low spirited, and inclined to melancholy. Where this is a prominent characteristic of the disease, no questioning will be necessary to bring the symptom out; it will be manifest upon all occasions. Here, carduus is the remedy for first choice Obscure pains in the pectoral region or other part of the thorax, as under the left scapula, would also suggest carduus.

A prominent indication for *ceanothus* is pain in the spleen. With marked splenic enlargement attended by much pain, it should have the preference over others of its class, as a rule, though there is no objection to rotation of the others where this fails. However, we are here prescribing something that is very direct, and there is little probability of failure if a proper diagnosis has been made.

Painless enlargement of the spleen may be taken as an indication for the use of *polymnia*. And here it is well to make avail of the external, as well as the internal, use of the drug. The polymnia ointment here comes into use, it being applied over the enlarged organ, and rubbed in with plenty of friction. Dyspeptic complication of marked character is another indication for polymnia, such as burning in the stomach after eating, or fullness and distension with gases, attended by difficult, sighing respiration. This comes very near the condition characterized as "præcordial oppression," a symptom also suggesting polymnia in this disease, though not excluding the others, should this fail.

Grindelia squarrosa has not been tried as thoroughly as the three already canvassed. There may be obscure cases, not very well marked, where this will do better than any other remedy. It has relieved splenic pain and dyspeptic symptoms of long standing for me very satisfactorily in several cases, and I should expect much from it in any case of splenic enlargement in malarial cachexia. It needs more study to fix a place for it. As we do not object to a reasonable amount of combination, two, three, or all these remedies may be combined occasionally, though usually I would not expect as prompt results as where a proper selection had been made of one or two of them.

A natural sequence of splenic congestion is hepatic disturbance,

of greater or less severity. Sometimes this amounts to congestion of the liver, announced by enlargement, with tenderness on pressure. In other cases, there may only be functional inactivity. In either case icteric symptoms are likely to be more or less manifest. Congestive hepatic disturbance will suggest the use of *chelidonium*, in combination with the appropriate spleen remedy; hepatic torpor without congestion will properly be met with *chionanthus*. Chelidonium should not be given in more than two- or three-drop doses, repeated four times daily; chionanthus may be given in ten-drop doses, at about the same intervals. A good method would be to combine the spleen and liver remedies in the same dose.

Often the *stomach* will be found to give rise to the most prominent symptoms. We have the torpid stomach, where the tongue is heavily coated at the base, and where there seems to be a morbid accumulation in the viscus, as suggested by eructation of gases, difficult digestion, etc. When this condition is present it will be found a persistent one, and its removal will become an important matter. The treatment here is the administration of *emetics*, repeated once or twice a week, until the eructations have ceased, and the tongue has assumed a normal condition. I usually employ powdered *ipecac*, giving from three to five grains, stirred in half a cup of hot water, repeating every ten minutes, until free emesis follows. It is well to order an extra teacupful of hot water between the doses. In other cases, the tongue will be broad and flabby, and evenly coated with a dirty, pasty white coating. This is also a persistent symptom, and must be removed, in order that the patient may make rapid progress. We expect to correct this with *sulphite of sodium*; dose, a grain, in capsule, four times daily, continued until the coating has disappeared. Gastric irritation is another symptom that sometimes demands attention, though it is not common in malarial cachexia. It is recognized by the pointed tongue, with reddened tip and edges, accompanied by vomiting, and disgust for food. We remedy this by using, for several days, a mixture of five or eight drops of *aconite* with twenty drops of *rhus tox.*, in four ounces of water; dose, a teaspoonful every one or two hours. Meantime, our spleen remedies should be steadily administered, whatever other treatment may be indicated.

Normal activity of the general circulation is an important consideration, whatever the condition may be. Even splenic congestion may be reached by a method which will provide for a vigorous cutaneous capillary circulation. The *vapor bath*, either the alcoholic, or, what is preferable, the steam cabinet bath, is an admirable remedy in chronic malarial poisoning. It very materially assists, and is even

capable of curing most cases, unaided by other means. When practicable—where the means are at hand—a steam cabinet bath may be taken every other day for a fortnight, and afterward twice a week, until recovery has been fully established. This does not interfere with other treatment and is a powerful adjuvant, to say the least of it. The gastric, hepatic, and splenic symptoms yield to it speedily, and digestion and assimilation are speedily restored to a normal condition. The baths may be much aided by using, in conjunction with them, the *tonic faradic* treatment described in Dynamical Therapeutics.

Some authors advise the removal of the patient to a non-malarious district. This is good advice, but, unfortunately, a large percentage of these patients are not financially able to incur the expense of such a change. We must be able to do better than choose one of these alternatives—sending them away, or leaving them at home to die.

We do not want much quinine in the treatment of these cases. We will find, if we make the trial, that they have worn quinine out—that this drug aggravates instead of ameliorating. If there is periodicity and quinine is administered for its interruption, no impression will be made, usually. It is not impossible that malarial cachexia may really be largely chronic quinine poisoning. At least that drug bears the reputation of producing portal congestion, and in this case such a condition is just what we are endeavoring to get rid of. *Alstonia* and *arseniate of quinia*, in alternation, are more effective here, and less objectionable. As a steady tonic here, when I desire to employ an anti-malarial agent, I prescribe the following capsule: ℞ Alstonia constricta gr. iii, arseniate of quinia, 3x trit., gr. ii. M., and fill one capsule. Duplicate No. 30. S, One after each meal.

Since the foregoing was written, I have given especial attention to *grindelia squarrosa*, as a general curative agent in chronic malarial cachexia. A careful review of Prof. Bundy's writings, and an investigation of some of his arguments as to unreliability of preparations of this agent often found in the market, with subsequent extended use of a tincture prepared by myself from the recent plant, have convinced me that this is the best remedy we possess for the cure of malarial cachexia. I will premise quotation of what Prof. Bundy has written upon the subject, by asserting it as my belief, that he has not overestimated the value of this remedy, and that when a reliable preparation—one true to name—is employed, it will seldom prove disappointing. Following is the quotation with reference to the subject, from Prof. Bundy's pen:

"The continuance of chronic intermittents is most frequently the

result of splenic hypertrophy. The hypertrophy is a secondary matter at first, but when well established it becomes the perpetuating cause. This, I am satisfied, is a fact, and so long as the hypertrophy exists, so long will the paroxysms continue to return. Quinine, arsenic, picrate of ammonium, etc., are given separately and combined in every conceivable manner, yet the paroxysms appear every eighth, fifteenth, or twenty-second day for months, until the patient becomes bloodless and reduced to a mere skeleton, the abdomen distended by an enlarged spleen, and from dropsical effusion.

"What is necessary in this case is to remove the splenic hypertrophy, which is positively the perpetuating cause, in combination with malarial influences, if the patient lives in a malarious district. 'There is a balm in Gilead,' and when the profession has frittered away time enough in 'tinkering' with routine and hackneyed treatment in unsuccessful attempts to cure chronic intermittents, it may see fit to resort to this remedy, and learn how to succeed in curing them.

"The drug is *grindelia squarrosa*. I have cured over seventy cases in the past four years, and I have yet to see the case it will not cure if properly given. That it may sometimes fail is entirely possible, as almost any remedy is liable to sometimes disappoint; but failure in my hands in curing chronic ague has never occurred with this remedy.

"My last case is that of a child 18 months old. The spleen was four times the natural size; and this condition had existed seven months when I commenced treatment. I gave ℞ Fluid extract of grindelia squarrosa (P., D. & Co.'s) ʒiii, syr. acacia and aqua dest. aa, ʒii. Sig., a teaspoonful four times daily. ℞ Tinct. ferri. chlor. ʒi, simple syrup ʒiii. M. Sig., one teaspoonful four times daily. One prescription was given before and the other after meals. From three to ten weeks of this treatment may be necessary."

Bundy believed that there was a great deal of fluid extract of grindelia squarrosa in the drug market which was not true to name, but which was, instead, fluid extract of grindelia robusta. For the two varieties resemble each other very much, and are liable to be confounded by inexperienced herb-gatherers. This may explain the failures which often attend the use of this remedy in the treatment of chronic malaria. From extended personal experience with a tincture prepared by myself from the recent herb gathered near Colusa, by a medical gentleman who knew Bundy while there, I am convinced that the fluid extract of Parke, Davis & Co., labeled grindelia squarrosa, is true to name, and I am doubtful about the identity of every other preparation I have found in the market.

XXI. ANTHRAX.

Synonyms.—Malignant Pustule; Splenic Fever; Malignant Œdema; Wool-sorter's Disease.

Definition.—An acute, infectious disease, caused by the bacillus anthracis, characterized by destructive inflammation at the seat of inoculation, and severe constitutional symptoms of grave character, arising therefrom.

Etiology.—The *bacillus anthracis*, the active principle of this disease, was the first specific microbe of disease detected and described. It is from two to three times the diameter of a red blood-corpuscle in length, the rods often being united. It multiplies by fission, and grows very rapidly on culture medium, the spores possessing remarkable vitality, though the bacilli are easily destroyed. It is introduced into animal tissues by inoculation from the bites or stings of insects, into the stomach with the food, and into the lungs through inspired air.

BACILLUS ANTHRACIS (FROM SMITH—FLEMING)
WITH AND WITHOUT SPORES. O.L. JONES DEL.

It is a malady principally infecting cattle and sheep, though those of the human family who are about and exposed to infected animals and animal products, are most liable to become diseased. Thus, butchers, tanners, wool-sorters, herdsmen, etc., are more liable to acquire the disease than ordinary individuals.

It is more common in certain parts of Europe and Asia than in this country, though it is not uncommon in South American cattle districts.

When a region becomes infected, the bacillus seems to be perpetuated for a long time. Pasteur believed that the bacilli might be brought from the buried carcass of an infected animal to the surface by earth-worms, and there prove a source of infection to new individuals. Others doubt the soundness of these views, though they admit the persistency of the infection, in regions once contaminated.

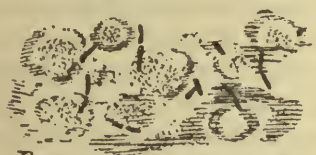
It will be described under the following heads:

External Anthrax.

Malignant Pustule.—In this form of anthrax, the inoculation usually occurs upon an exposed part, such as the face, hands, arms, or neck, and is probably due to the bites or stings of insects. The first announcement of the disease is a small papule, though its

appearance may be heralded a few hours by burning and itching in the vicinity. The papule enlarges rapidly, until a vesicle appears upon its summit and a hardened areola surrounds its base. Within thirty-six hours a brown slough appears in the center, denoting the point of inoculation. Numerous vesicles now appear, surrounding the vicinity of this point, and resembling the numerous openings of a carbuncle. The local inflammation spreads rapidly, extreme induration and swelling speedily appearing, and the lymphatics convey the irritation to neighboring glands, which become swollen, indurated, and painful. Constitutional symptoms soon appear, a rapid rise of temperature and other febrile phenomena being afterward followed by subnormal temperature, coma, and death. In favorable cases, the constitutional symptoms are not so severe, and the local symptoms are marked by sloughing at the point of irritation, with gradual healing of the cavity.

Malignant Œdema.—This occurs in parts containing a large amount of connective tissue, and which are liable to puffy swelling under other circumstances, such as the eyelid, head, hand, arm, and other parts. There is here an absence of the papule and vesicle, and, when sloughing occurs, a large surface is involved. The œdema spreads rapidly, involving large areas, and the constitutional symptoms are almost always extreme, and usually lead to a fatal termination.


Bacillus of Malignan. Oedema, very high power.

The character of the lesion—its progress—and the severe constitutional symptoms which attend, in addition to the occupation of the patient, will usually afford a clear diagnosis, in either malignant pustule or malignant œdema. Microscopical examination of the fluid of the affected part will disclose the presence of the anthrax bacilli.

Internal Anthrax.

Mycosis Intestinalis.—This form arises from the reception of the parasite into the stomach with the food, when the flesh of diseased animals is eaten, or the milk of affected cows is drunk. The symptoms are ushered in with a chill, and attended by severe gastro-intestinal disturbance, such as vomiting, diarrhœa, burning pain in the stomach and intestines, dyspnœa, cyanosis, and extreme restlessness, followed by coma or convulsions. This form of the disease usually attacks several persons simultaneously—those who have eaten, at the same time, of the flesh of an animal affected with anthrax.

Wool-Sorter's Disease.—This disease arises among the employés of large wool- or hair-sorting establishments, the microbe being inhaled during the handling of the diseased material. The hair and wool imported into Europe from South America and Russia seem to be most commonly infected. The dust arising during the handling of these products seems to contain the bacillus anthracis, at least the microbe gains entrance to the interior of the body during the commotion, either by way of the stomach or lungs, and serious symptoms follow. The patient is attacked with a chill, followed by fever, attended by racking pains in the limbs and back, short, rapid breathing, with severe pains in the chest, and usually signs of bronchitis. Gastric symptoms often supervene, vomiting and purging attending, and rapid prostration follows. Sometimes there is delirium during the advanced stage of the disease, and sometimes coma.

The diagnosis in such cases is difficult, and can only be inferred from the occupation of the sufferer and the violent symptoms, until investigated from a bacteriological standpoint.

Treatment.—The treatment thus far followed has not yielded a great amount of satisfaction. It is advised to destroy the pustule by the use of actual cautery or caustic potash, and sprinkle powdered bichloride of mercury over the surface. Subcutaneous injections of carbolic acid about the pustule, to prevent the spread of the bacilli, are also advised. It seems to me that such treatment cannot but make a bad matter worse, and aggravate the whole affair. Probably *echinacea* will be a better remedy, both internally and locally, the system being saturated with it. *Lachesis* and *crotalus hor.* would be applicable, also. It is a fact that heroic treatment often aggravates diseases which it is intended to relieve, and this is likely to be one of the instances.

XXII. HYDROPHOBIA.

Synonyms.—Rabies; Lyssa.

Definition.—Hydrophobia in man is an acute, specific, contagious, almost invariably fatal disease, due to an unknown microörganism, and transmitted by some animal, by inoculation. After a period of incubation, there are violent spasms of the muscles of deglutition and respiration; later, general convulsions, great prostration, and finally ascending paralysis, and death.

Etiology.—The disease is principally noticed in dogs (90 %), although it has occurred in cats, wolves, foxes, badgers, martens, hyænas, jackals, polecats, horses, asses, mules, oxen, and sheep.

The disease is common in Russia, less so in England and France, and rare in Germany and America.

It is produced by the bites of animals suffering from rabies, or by accidental inoculation of wounds with their saliva or blood, the latter occurring occasionally in autopsies of infected animals. The virus may be swallowed, or may come in contact with the unbroken skin, without developing the disease. It retains its vitality some time after the death of the animal. Communication from man to man is denied. The consensus of opinion seems to be against the spontaneous origin of rabies among animals.

All persons bitten by rabid animals do not contract the disease, the percentage of cases being about 12 to 14. The saliva is often prevented from gaining entrance to the wound by the clothing. In other cases, cauterizing the bite destroys the virus. Efforts to discover the microbe which produces the disease, have thus far failed.

Pathology.—There are no well-defined pathological changes. Rigor mortis is marked, and decomposition sets in early. The blood is thin, and darker than usual. The blood-vessels are more or less congested, excepting those of the heart, spleen, and liver, which are normal, as a rule. The pharynx and fauces are much congested, the same condition being observed in the mucous membrane of the alimentary and respiratory tracts. The brain and spinal cord, with their membranes, are hyperæmic, and considerable œdema is present. Scattered throughout the whole central nervous system, but more noticeable in the base of the brain and spinal cord, there are patches of inflammatory deposit.

Symptoms.—The stage of *incubation* is much longer than in most of the infectious diseases, and usually lasts from two to six weeks; rarely, it is protracted to a year or more. The wound heals, if sufficient time elapses, and the patient's health is about as usual. The length of this stage is influenced by the age of the individual, children manifesting symptoms of rabies sooner than adults.

From one to three days before the serious symptoms develop, the patient has more or less constitutional disturbance. There is headache, anorexia, insomnia, a feeling of dread and apprehension, and a general hyperæsthesia. The countenance has a look of anxiety, and the pupils are dilated. There may be pain in the region of the wound, with more or less tenderness and congestion, with occasional suppuration. Frequently, all local symptoms are absent. We now notice more or less respiratory oppression. There is difficulty in enunciation and deglutition, caused by muscular spasm, and a sensation of choking about the pharynx. Water is refused, on account of the painful spasmodic condition excited in the throat. There is an abundant secretion of viscid saliva, which cannot be swallowed, and is constantly expectorated.

Respiratory or pharyngeal phenomena may be the first indication of the approaching attack, other premonitory symptoms being absent.

The spasmodic condition at length begins to assume a serious phase, and the patient is excited, not only by attempts to swallow, but the sight of water, or a mere suggestion of drinking, producing a convulsion. General hyperæsthesia develops to such a degree that loud sounds, an unexpected touch, currents of air, bright lights, or the contact of the bedclothing, is sufficient to initiate a spasm. These seizures last from one- to three-quarters of an hour, and leave the sufferer exhausted. In the intervals, his mind is clear, but he is tortured by apprehension of returning attacks, and anticipations of his terrible sufferings. The convulsive action that was first confined to the respiratory organs, soon becomes general, with periods of hallucination and mania, due to excitement and partial asphyxiation. The mouth and fauces are dry, and there is a constant hawking of adhesive mucus and saliva, which are expectorated indiscriminately over the bed and attendants. The pulse, which was at first normal, grows weak and rapid, and the temperature rises, raging from 101° to 103° F. This stage of the disease lasts from one to three days, and death may occur from exhaustion or asphyxia.

Occasionally there are no convulsions, the patient complaining principally of dyspnœa. Some few are enabled to swallow throughout the disease, although considerable pain accompanies deglutition. There is now a gradual transition to the third stage, the stage of *paralysis*, which lasts from three to eighteen hours. The prostration grows more marked, and there is a diminution of convulsive action. Respiration is much easier, and the spasmodic condition of the laryngeal muscles is diminished, so that liquids can be swallowed. Rapidly ascending paralysis commences, and respiratory and cardiac failure closes the scene.

Diagnosis.—After hydrophobia has fully developed, there is no possibility of a mistake in diagnosis. The only diseases that may simulate it are lyssophobia (hydrophobia imaginary), tetanus, and cases of epilepsy or hysteria, where the organs of deglutition are affected. In rabies, the muscles of mastication are not affected, the convulsions are not tonic, and the apnœa is due to spasms of the laryngeal muscles and not to those of the chest. Hysterical persons sometimes develop symptoms that simulate hydrophobia. They imagine they have the disease, and have paroxysms, in which they refuse to drink, grasp the throat, and manifest more or less violence in their excitement. There is no elevation of temperature, and the hallucination lasts longer than true rabies. Probably in these cases hypnotic suggestion could be successfully used.

Treatment.—The disease once established, death is almost inevitable, and we can only use such methods as will modify the severity of the dying struggle.

Prophylaxis, therefore, assumes great importance, and all suspicious cases should receive prompt attention. The physician is not called, as a rule, until some time after the injury, and the destruction of the lacerated flesh should not be delayed. If chloride of zinc, caustic potash, or concentrated carbolic acid are at hand, the wound should be cauterized, after being carefully washed. If there is likely to be any delay in procuring the cauterizing agent, the flesh that has come in contact with the teeth should be excised, the knife cutting wide of the wound. It is advisable to apply a cup, and favor hemorrhage. The actual cautery, while severe, is efficient. The wound should be kept open for at least a month. If there is any doubt about the dog having hydrophobia, it should be kept penned up a sufficient time to develop the disease. When the animal is not mad, the patient may thus be saved weeks of useless apprehension and mental suffering.

The discovery of *preventive inoculation*, by Pasteur, created quite a furor, and institutions for the treatment of rabies have been founded in various parts of the Old World. Pasteur found that the virus increased in potency when a number of rabbits were successively inoculated, so that when this more virulent product was used, only seven days elapsed before the symptoms of hydrophobia developed, whereas fifteen were required in the primary inoculation. The virus is taken from the spinal cord of the rabbit, and it was discovered that it gradually decreased in intensity when the cords were preserved in dry air. A dog was now inoculated with virus twelve or fifteen days old, and the process was repeated with stronger solutions, until it was found that he had acquired immunity from the disease, and that the most potent virus had no effect upon him. In his treatment of patients, Pasteur used virus of greater intensity on successive days, and claimed to be able to abort the disease when the patient was seen early enough. Much doubt exists, however, with regard to the efficacy of Pasteur's methods.

In former times, the unfortunate who developed rabies was smothered between two mattresses; and, although the practice seems barbarous, it was undoubtedly humane.

All treatment fails to check the course of the disease, so our attention is directed toward alleviating the suffering of the patient. He should be removed, by his attendants, to a dark room. Chloroform anæsthesia, and narcosis by hypodermic injection of morphine, are carried to extreme length, all milder agents being discarded as

useless. *Cocaine*, applied to the throat, will diminish the hyperæsthesia, and permit of liquids being taken. Death occurs in from two to ten days. In rare cases, patients may survive for three weeks. *Curare*, *amyl nitrite*, and *nitro-glycerine*, recommended in the article on tetanus, may be tried, but reported cures are open to suspicion.

Professor Goss asserts that *echinacea* exerts a prophylactic influence, when administered steadily after the period of inoculation, in fifteen- or twenty-drop doses, repeated four or five times daily.

Cures have been reported from the inhalation of *oxygen gas*, after the spasmodic symptoms have appeared.

XXIII. TETANUS.

Synonyms.—Lock-jaw; Trismus.

Definition.—Tetanus is an acute or chronic infectious disease, characterized by a progressive tonic spasm of the voluntary muscles, with paroxysmal exacerbations, resulting, as a rule, in death. It may occur in epidemic form among the wounded, in times of war, or in children, in lying-in hospitals.

Pathology.—Where pathological lesions are present, they are secondary, and dependent on the excessive muscular spasm, the primary disturbance being reflex and functional. Occasionally the nerves supplying the affected parts show inflammatory changes. The spinal cord and medulla are at times hyperæmic, and there may be effusions and more or less extravasation.

Etiology.—Tetanus is due to the bacillus tetani, which gains an entrance into the body through a traumatism. The old division of the disease into traumatic and idiopathic tetanus is questioned by modern authorities, and all cases are believed to be due to an injury of some character.

BACILLUS TETANI. COVER GLASS CULTURE.

The *bacillus tetani* is short and straight, with an enlargement at one end, due to sporulation. Several ptomaïnes are derived from it, and it is believed that the irritation of the nervous system is mainly due to their presence, as but few of the bacilli are to be seen in the body. The spores are to be found in manure, garden soil, decomposing liquids, masonry, and the dust of streets. It is anaërobic.

Tetanus may follow injuries of most any kind, such as wounds, burns, fractures, or dislocations. It has occurred after abortion, and normal labor. Surgical operations, such as the ligation of piles, amputations, castration, or even the passage of uterine sounds, have

been followed by lock-jaw. In infants, the bacilli gain an entrance through the navel.

A rare case has been reported, where the disease was due to an accumulation of bird-shot in the appendix vermiformis. Where tetanus follows a simple fracture or dislocation, the disease is supposed to be due to internal infection. Telluric conditions are believed to have much to do with the development of tetanus. It is more frequently met with in hot climates. It has been frequently noticed that, after battles, sudden changes of temperature have been followed by the development of many cases of this disease among the wounded. While no age or sex is exempt, adult males furnish the greater number of cases.

Symptoms.—The stage of *incubation* is variable in length, but it generally lasts about a week.

The patient first notices a stiffness in the neck and lower jaw, and is apt to attribute it to a cold. As the symptoms become more noticable, there is difficulty in mastication and deglutition, and these acts are attended with more or less pain. The lower jaw becomes fixed, as the depressors are unable to overcome the spastic contraction of the temporal and masseter muscles. The face becomes distorted, the muscles of expression contracting, and producing the characteristic *risus sardonicus*. The spasmodic condition gradually involves the other groups of muscles, the wrists and fingers being the only parts of the body not sharing in the general and gradually increasing contraction. The trunk becomes rigid, and, on movement of the diaphragm, a severe pain passes from the ensiform cartilage through the body, accompanied by a distressing dyspnœa. The lower extremities are in most cases in a line with the body, the head is drawn back and fixed, and the arms either parallel with the trunk or drawn across the chest. The abdomen is is hard and broad. In general, the powerful muscles of the back and limbs bind the body in the form of a bow, and, during exacerbation, the patient rests upon the occiput and heels. This position is termed "opisthotonos." More rarely the spine is bent forward and the head cōmes in contact with the knees, a condition of "emprosthotonos." Still more uncommon is "pleurosthotonos," the muscles of one side of the body giving a lateral curvature to the spinal column. The muscular spasm is continuous, but there are paroxysmal exaggerations, so severe as to, in some cases, project the patient from bed. These convulsive seizures have been known to fracture a bone or rupture a muscle. They are excited by attempt at muscular action, or are spontaneous. Soon, however, sudden noises or any disturbance will produce them, and the patient lies in constant fear of another seizure.

During the interval when the cramping pain ceases, there is soreness and aching in the muscles. The bowels are almost always constipated. Frequently, contraction of the sphincter muscle of the bladder causes retention of urine. Here priapism will often be noticed. The body is wet with a profuse perspiration. The mind remains unclouded, and the patient retains his faculties to the last. As the result of the excessive muscular contraction, the pulse and temperature are more or less affected. Frequently, just before death, the temperature rises as high as 114° F. As food cannot be taken, and rest is impossible, exhaustion begins early, the patient lasting only two or three days. Respiration is greatly embarrassed during the spasms, and death often occurs from apnœa.

Chronic Tetanus: Tetanus Mitis.—In chronic tetanus there is a much longer period of incubation. Rarely, an acute attack may assume a chronic type, and, if so, there is a possibility of recovery. In tetanus mitis, the muscular involvement is extensive, but there are intervals when there is a partial cessation of the spasm. In favorable cases, these, growing longer, permit of the patient's obtaining some rest. Some few of these cases recover. A mild form of tetanus, where the muscles of the neck and face are alone involved, is termed "trismus."

Diagnosis.—From cerebral or cerebro-spinal inflammation, by there being no coma or delirium, and the absence of fever during the intervals of the attacks.

In strychnia poisoning, consciousness is lost, the muscles of the neck, head, and jaw are not primarily affected, there is retinal hyperæsthesia, and objects look green. The vomitus, when analyzed, will give the strychnia reaction. Hysteria or epilepsy may slightly resemble tetanus, but only during its earlier stages. In its milder forms, it has been mistaken for rheumatic inflammation of the jaws.

Prognosis.—The prognosis is grave, especially in wounds received in battle. Chronic cases occasionally recover. After the fifth day, there is a fighting chance, and when the patient passes the twelfth day, the prognosis is quite hopeful. The disease is invariably fatal in the very young, where the period of incubation is short, and when rigidity begins early.

Treatment.—Prophylaxis.—All suspicious injuries should be disinfected. Where the wound has become foul, or when it has been produced by some dirty object, especial care should be taken to render it aseptic. Foreign bodies should be sought for if there is a possibility of their having found lodgment in the tissues. In epidemics, all cases should be isolated.

Management.—As in rabies, the patient should be placed in a

darkened room, and kept absolutely quiet. All noises, and everything of a disquieting nature, should be prevented. *Chloroform* is administered by inhalation, frequently. *Chloral* by rectum, and *morphine* subcutaneously, are administered in large doses, milder acting drugs being useless. *Potassium bromide*, ʒi every three hours, is highly recommended. It may be combined with the chloral. The rigidity of the muscles may be partially overcome by deep hypodermic injections of *atropine*. *Amyl nitrite* and *glonoin*, theoretically, should help to relieve the spasm, and have been used more or less successfully, in tetanus. As we desire to disturb the patient as little as possible, remedies that may be administered by inhalation, subcutaneously, or by rectum, are of prime importance. *Curare*, being a motor paralyzer, is indicated, and may be pushed to its full physiological influence. Of the crude drug, we may administer from gr. 1-20th to 1-5th. *Curarine* may be used hypodermically, in doses of from 1-200th to 1-100th of a grain.

A valuable remedy, and one which may be obtained almost anywhere, is *tobacco*. The officinal infusion (ʒi–Oi) may be given by enema, ʒiv being the maximum dose. In administering, we regulate the dose and time of repetition by the effects produced. It should be used cautiously. It is absorbed very rapidly by the stomach, the alkaloid being given in minute doses. If desirable to administer hypodermically, the following formula may be used: ℞ Nicotine gr. ss., aqua dest. ʒii. M. Sig. 1–24th gr.

Hale asserts that *passiflora incarnata* is a cure. It is probably not superior to gelsemium and scutellaria, which, while of benefit in trismus, fails in tetanus. Where there is retention of urine, the catheter should be used. Highly nutritious foods should be given, either through a stomach tube or by enemata. Success has been reported with injections of the tetanus anti-toxin of Tizzoni and Cattani.

But we cannot expect to cure tetanus with specifics in many instances. When the period of incubation is long-continued, as frequently occurs, we will find that the general system has taken on a septic condition, which must be corrected before anti-spasmodics will afford much satisfaction. Each individual case will require careful analysis, and where the best anti-spasmodic may fail, the properly directed antiseptic treatment may promise much. I have in recollection a severe case of trismus, which occurred years ago, that seemed to be rapidly passing into a hopeless stage, in the hands of an allopathic physician, which rapidly improved, and nearly recovered within a week, when alcoholic *vapor baths* were administered every twenty-four hours, and their action aided by the internal administra-

tion of *sulphite of sodium* and *baptisia*, the former drug being prominently indicated by the pasty-white coating on the tongue.

In malarial regions, periodicity may be a marked feature of the disease, the spasms becoming violent during the exacerbations, and almost, or quite, disappearing during the remissions. Here we must exhibit *quinine*, in antiperiodic doses, promptly, if we are to expect benefit from other treatment.

The physio-medicalists expect the steam bath to accomplish much toward a cure, and this, aided by such relaxants as *lobelia, scutellaria, gelsemium*, etc., constitutes a very effective means of treatment, these practitioners being very successful here.

Aplopappus laricifolius, if the fresh plant can be obtained, is an excellent remedy, though whether much can be done with a tincture without a fomentation of the plant to the affected part, remains to be proven by experience. From experience with it in veterinary practice, upon my own carriage horse, I am not favorably impressed with it.

XXIV. ACUTE GENERAL TUBERCULOSIS.

Synonyms.—Acute Miliary Tuberculosis; Typhoid Tuberculosis.

Definition.—An acute, infectious disease, most common to the period of puberty, characterized by the rapid dissemination of tubercles throughout the entire body by auto-infection, the tubercles being usually concentrated in some vulnerable portion, such as the lungs, the mesenteric glands, or the meninges, with almost invariably rapid and fatal termination.

Etiology.—An understanding of the etiology of this disease necessitates a study of that of tuberculosis in general, as acute general tuberculosis is but a variety of a disease which manifests itself in various phases. For many years the study of tubercle has been attended by much obscurity and dissatisfaction. The doctrine was long adhered to that tubercular deposit was a result of inflammatory action, it being due to some peculiar predisposition of the system—to a dyscrasia. This idea has, in recent years, been well proven a fallacy, and the individuality of the disease—its tangible identity—pointed out. Tubercle is now believed, by modern pathologists, to arise from the destructive action of the tubercle bacillus, a parasitic microörganism, the discovery of which was announced by Koch, in 1882.

The *tubercle bacillus* is a slender rod, which is about one-third the diameter of a red blood-corpuscle in length, and about five times as long as broad. It may be straight or slightly curved, as seen under

the microscope, uniform in appearance throughout, except that certain individuals exhibit from four to six highly refractive spherical spaces along the body, at regular intervals, which are supposed to represent spores. These seem to be particularly numerous when tubercular disease is developing rapidly, while in cases which are quiescent, or retrograding, the spores are absent. The bacilli manifest a remarkable resistance to destructive agencies, and retain their vitality almost indefinitely, even resisting the bleaching action of acids, when once stained in the bacteriological laboratory.

Bacilli may supposedly be expectorated in tuberculous material, and become a part of the common dust, by desiccation and exposure, to afterward enter the lungs of uncontaminated individuals through inspiration, during a disturbance in the atmosphere, and produce fatal infection, provided the subjects are susceptible.

It is not difficult to explain many of the seeming inconsistencies of the theory of the bacillus-origin of tuberculosis. It has long been a recognized theory that the disease, or predisposition to it, is hereditary. This doctrine need not be greatly disturbed by the new pathology. Some individuals seem remarkably susceptible to the disease, while others seem proof against it, and this receptivity tends to run in families, though not so confined, by any means. A consumptive wife may infect her husband, while perishing from the disease, and *vice versa*, the activity of the disease-agency remaining dormant in the second individual for years, to afterward develop an activity fatal to that individual, and, possibly, to others intimately associated. People thrown much together are liable to communicate it to one another. Houses in which consumptive families have resided, seem to retain the infection for years, and those of the most perfect physique may develop the disease from occupying them afterward, though they may remain the hosts of the bacilli for years before active disease becomes manifest. Public institutions, especially penitentiaries, where close confinement is the rule, seem to breed the infection. It is a notorious fact that tuberculosis is very common in these institutions, and that the most robust and hardy con-

stitutions succumb to it after a few years' confinement, where it has been breeding for a long time. The disease has even been conveyed from a consumptive mother, through the placenta, to the child in utero.

Diet is not an uncommon source of contamination. The tubercle bacillus thrives in other animals than man, especially in bovines. Cow's milk may therefore be contaminated from within, and may become a prolific source of the disease. The custom which has prevailed within the past years so largely of raising children on condensed milk, has doubtless much to do with the presence of miliary tuberculosis at the period of adolescence, the activities which are then aroused in the organism assisting in the rapid distribution of the bacilli and their speedy destructive action. Doubtless there are some who resist the infection when exposed, and escape altogether, while others may become hosts of the parasites and resist them sufficiently to reach adult life, before some predisposing accident places the constitution in a sufficiently depraved condition for the bacilli to accomplish their ravages.

Pathology.—In acute miliary tuberculosis, the bacilli may be distributed from any center where they first become lodged, through the lymphatics, veins, and even the arteries, becoming disseminated through all the tissues of the body, except, perhaps, those of the salivary glands and pancreas (though it is now asserted that the last-named organ is not exempt).

The lungs are most frequently and largely involved, then the liver, intestines, kidneys, spleen, pia mater, peritoneum, pleura, dura mater, and brain. Deposits are more rarely and sparsely distributed in the thyroid gland, suprarenal bodies, female genitals, striped muscles, and stomach.

When the lungs are infiltrated, the condition is easily recognized by the eye upon autopsy. The organs are filled with little gray transparent nodules of varying size, some being so small as to be hardly noticeable, while others are of the size of a pin's head, or a millet seed. If a portion of the lung be taken between the finger and thumb, it imparts a hardened, shotty sensation, and, if the lung be sliced, lumpy elevations, corresponding with these bodies, may be observed upon the freshened surface. They are usually transparent, though some of them may have an opaque center, suggesting the commencement of gaseous changes. Inflammatory changes are also more or less marked in the lung tissues, such as œdema, catarrh of the mucous membrane, plastic exudation, etc. The pleura may be involved, and found to be the seat of more or less tubercular deposit, as well as of former inflammatory action. Sometimes the tuberculi-

zation is confined to a portion (as a single lobe) of the lung. The liver and spleen present similar appearances when notably affected, the tubercles being quite evenly distributed through the organs, and showing a slight tendency to coalesce.

Tubercles show a strong tendency to caseation, in most instances, but this disease runs its course so rapidly that little change in this direction occurs. In all fresh tubercles, bacilli are found upon microscopic examination, but the sputum does not contain these microörganisms in this affection, as they do not break down before death. When the tubercle has gone on to necrosis and caseation, they are not so abundant, only the spores remaining in the cheesy detritus.

Symptoms.—A prominent symptom is fever, usually ushered in with a chill, or a succession of chills. The fever is irregular, the temperature running from 102° to 104°, the skin being hot and pungent, sometimes dry, and sometimes bathed in a sticky perspiration, the pulse being remarkably feeble and rapid, the tongue pointed, and reddened at the tip and edges, or dry, brown, and fissured, the urine scanty and high colored and the bowels constipated, unless there is intestinal irritation.

Soon there is a hectic flush on the cheek, the skin becomes transparent, with prominent, superficial veins; rapid emaciation follows, with extreme prostration. Cutaneous eruptions, such as sudamina, roseolous rash on the chest and abdomen, and herpes labialis, are not infrequent accompaniments.

A remarkable feature of most cases is the pulmonary irritation, manifested by dry, hacking cough, with succeeding expectoration, muco-purulent, at first, and, later, sanguinous in character. With these symptoms are remarkable increase in the number of respirations (50 or 60 to the minute) with dyspnœa, and cyanotic expression of countenance. Sibilant and subcrepitant râles now abound, and areas of dullness, with bronchial breathing, are found later on.

When the meninges are principally affected, there is intense headache, photophobia, extreme restlessness, delirium, facial palsies, stupor, convulsions, coma, and Cheyene-Stokes breathing. Tubercles may occur upon the retina, with attending visual defects.

When the intestine and peritoneum are the principal points of deposit of the tubercles, there are pain, tenderness, abdominal fullness, diarrhœa, and, often, gastric irritability.

The disease may last five or six weeks, though it usually terminates fatally in from two to four. Death most frequently results from pulmonary œdema and asphyxia, or cerebral anæmia and collapse, though when the meninges are largely involved, convulsions, paralysis, or coma, may terminate the scene.

Diagnosis.—The irregular fever, the marked local symptoms, usually of pulmonary origin, the absence of epistaxis (a common symptom in the early part of typhoid fever) and markedly rapid respiration, with cyanotic symptoms, will distinguish this disease from typhoid fever, which it resembles somewhat, in its superficial aspects. It also resembles cerebro-spinal fever when the tubercles involve the meninges and brain, in some respects, though it runs a more rapid course than the slow form of that disease, and is not attended by the tonic spasms which mark the active form.

Prognosis.—The prognosis is almost invariably fatal. All that can be expected of treatment is to palliate the most unpleasant symptoms, and render the last hours of the patient as endurable as possible.

Treatment.—This must be unsatisfactory, at best. There must necessarily be a steady progress from bad to worse, and the treatment which may succeed in palliating to-day, will naturally lose its effect to-morrow, seeing that the disease is steadily progressing toward a fatal termination, and that the structural changes are continually more and more aggravated. However, we may lessen the severity of some of the unpleasant symptoms, and do this in the beginning without the use of opiates to any great extent, though toward the close of the disease opiates are about all that will afford any relief from the cough and other unpleasant symptoms.

Gastric irritation will be a common cause of complaint, nausea and disgust for food, being a common feature. To relieve this, as well as to control the fever and restlessness, a combination of *aconite* and *rhus tox.* will be found excellent. Add five drops of aconite (Lloyd's or Worden's) and ten or fifteen drops of a saturated tincture of fresh rhus tox. leaves to half a glass of water, and give a teaspoonful every hour. This excellent prescription will render good service for a long time, and even throughout the disease, lessening the fever, quieting nervous erythism, and alleviating pulmonary irritation, to considerable extent.

The *lungs* will usually demand something positively soothing, to lessen the tendency to continual hacking cough. It is best to avoid morphine internally here as long as possible, and the following prescription, used in the form of a spray, by inhalation, will serve a good purpose, for a time, at least. ℞ Essence of peppermint ʒi, aqua ℥i, morphia sul. gr. i, glycerine ʒi, carbolic acid gtt. xv. Mix, and use as often as required, three or four inhalations of the vapor being taken at a time.

Sometimes antifebrin will be excellent, to lessen the fever and soothe the pulmonary irritation, and there can be no objection to its

use here, as there can be no danger of after-effects, seeing that the case is hopeless at any rate. When these measures fail, the internal administration of *morphine* may be begun, to quiet unpleasant symptoms. *Codeine* is often preferable to morphine in alleviating cough and other unpleasant features, as it interferes less with the secretions, and is less irritating to the nervous system. It may be administered in syrup, in doses of from 1-4th grain to 2 grains.

To assist in alleviating the pungent heat of the surface and promoting rest, as well as to restrain the colliquative sweats which are liable to be present, a cool solution of *citric acid*, ʒi to the pint of water, may be used to sponge the surface, once or twice a day.

Stimulants, such as quinine, or whisky, should be avoided, as they can but increase the discomfort of the patient. Only sufficient food to supply the demands of hunger should be given, and plenty of cold water (as this will usually be craved) may be allowed, though iced water, when taken too freely, is liable to provoke abdominal pain.

During the last few days, it may be necessary to administer opiates freely, to control the cough and lessen the restlessness. Abdominal pain may demand *colocynth*, *dioscorea*, or *nux*, and diarrhœa may call for *bismuth* and *morphine*, in appropriate doses.

XXV. SYPHILIS.

Synonyms.—Pox; Lues Venerea.

Definition.—A specific, contagious disease, of venereal origin, of slow development and chronic course, which may be congenital or acquired, manifesting itself, when inoculated, by a series of pathological changes, which usually occur in regular order, as follows: First, a special tissue-change at the point of introduction, occurring from twenty-one to twenty-five days after inoculation (primary syphilis); second, constitutional symptoms, which develop within two or three months afterward, characterized by fever, cutaneous eruptions, irritation and ulceration of the mucous membranes, especially that of the pharynx (secondary syphilis); and third, granulomatous growths, which develop three, four, or five years afterward, affecting the muscles, bones, and skin (tertiary syphilis).

Historical Note.—Though doubtless a disease of greater antiquity, general attention was not called to syphilis as a peculiar and formidable disease, until the year 1494, when it occurred as an epidemic, among the troops of the French king, Charles VIII, who was then besieging Naples. From here, it seemed to spread all over Europe, and contemporary medical writers of various nations styled

it, according to caprice and prejudice, the "French disease;" the "Neapolitan disease;" the "Spanish disease;" the "German disease," etc. As this outbreak was contemporary with the return of Columbus' sailors from the voyage of American discovery, a popular belief arose that the malady had been transmited from the American Indians. It soon became apparent that the disease originated from sexual intercourse, and that crowded quarters, promiscuous and excessive indulgence, and indifference to slight venereal abrasions, promoted the spread of the affection in virulent form; and it is not unlikely that these circumstances favored its rapid spread and alarming prevalence, in Charles' army.

For nearly three centuries afterward, the profession universally confounded all forms of venereal disease with syphilis, and considered them of common origin. In 1767, Balfour declared that gonorrhœa was a separate disease, distinct from other forms of venereal disorder, and a local affection. These views were combated by the profession generally, however, and it was nearly thirty years later (1793) before another writer of distinction (Benjamin Bell) espoused this doctrine. Still the profession stood aloof from such views, and it finally remained for Ricord, thirty-eight years later (1831), to reiterate them, and convert the profession generally to their acceptance. Ricord, however, left chancroid and syphilis confounded, and it has only been within the past twenty years that the true distinctions, which enable us to classify the latter affection, have been fully established.

Etiology.—Doubtless syphilis is due to the presence of a specific germ, which causes all the pathological manifestations—where mercury does not aggravate its action. Several observers, notably Lustgarten, have observed bacilli in the secretions and morbid products of syphilitics, resembling the smegma bacillus, which they believe to be the active principle of the disease. Lustgarten always found them inclosed in round cells, probably the micrococci of other observers. Further study, however, seems necessary to firmly establish the identity of the microörganism of syphilis.

While contagious through inoculation among human beings, many assert that the disease is not communicable to the lower animals, others claiming that apes and monkeys are susceptible. One attack affords immunity from subsequent ones generally, and a mother who has borne a syphilitic infant seems protected from it, even though she may not manifest any evidence of having been affected as a result, the suckling and handling of syphilitic offspring producing no ill effect, while other wet nurses are readily contaminated.

Acquired syphilis is the result of inoculation with the blood or

morbid discharges of a person who has been comparatively recently syphilized. The longer the disease remains in the system, the less liability of contamination remains, as a general rule, though it may be communicated many years after the primary symptoms have disappeared, and healthy children may be begotten by parents recently syphilized. In from three to five years, however, the liability is almost entirely removed.

While inoculation is usually the result of impure sexual intercourse, there are many other avenues through which accidental infection may take place. The barber may transmit it with his combs, brushes, or razors; the dentist, with his lances or forceps; the physician, with his hypodermic syringe or thermometer; the surgeon, with his scalpel or other instrument; the gynæcologist, with his speculums, sounds, forceps, etc., and parturient women may infect the fingers and hands of midwives. Community drinking cups may become contaminated; kissing may communicate it; bestial practices often result in the communication of the disease to the lips and tongue, and, probably, insects and other pests, such as fleas, mosquitoes, etc., may convey it from one person to another. Humanized vaccine virus is quite an efficient means for the conveyance of syphilis, and, as the disease has thus been frequently spread by it, little use is made of humanized virus, though it is much more successful in transmitting kine-pox than virus from the bovine.

Hereditary transmission may be referred to either or both parents. Usually, a syphilitic husband or wife will infect the other parent before conception takes place, and it will be difficult to decide whether the child is syphilized through the sperm, or through the ovum. However, the male parent may impart the disease to the ovum without contaminating the mother, and the child be born syphilitic, while the only apparent influence exerted upon the mother may be that of rendering her immune against infection. Or, the mother may become infected, and the child may or may not be contaminated through the placenta.

General Pathology.—The *primary* sore (chancre) of syphilis is surrounded by a diffused infiltration of the connective tissue with small round epithelioid and giant cells, among which are found the bacilli of Lustgarten. Thickening of the intima of the small arteries, and alterations in the nerve-fibers distributed to the part, also occur. Hyperplasia and induration of the neighboring lymph-glands are associated with this condition. When the chancre is mixed with the virus of chancroid, rapid breaking down of these tissues occurs, the edges of the ulcer thus formed becoming raised and indurated, as the time for primary development arrives.

The lesions of *secondary syphilis* are many and diversified. There are ulceration of the fauces and irritation of the laryngeal mucous membrane (especially when mercury has been administered to excess), eruptions of various kinds on the skin, condylomata about the genitals, iritis and other eye affections, etc., these being attended, during the first two or three months, by protracted fever.

Tertiary syphilis is marked by syphilomata (gummata), which develop in the bones, periosteum (nodes), skin, muscles, lungs, liver, kidneys, brain, heart, testes, and adrenals. They differ in size, varying from very minute bodies, almost microscopic, to large, solid tumors, an inch or more in diameter, these being hard and resisting, except when they are located in the skin or mucous membranes; when breaking down, rapid ulceration may attend. Histologically, they consist of granulomatous tissue, resembling tubercle, a cross-section affording a grayish-white, homogeneous appearance, consisting of a periphery of translucent, fibrous tissue, with a firm, caseous center. Dense, sclerotic tissue may envelop clusters of three or more of these bodies. As few blood-vessels are supplied to these bodies, there is a constant tendency to breaking down of the central portion, by coagulation necrosis and the formation of fibro-caseous material, while progressive fibrous growth occurs at the periphery. Absorption of the caseous material may ultimately result, a fibrous scar remaining.

Acquired Syphilis.

Pathology and Symptoms.—The period of *incubation* varies from two to six weeks, though it usually lasts about four. During this time there are no symptoms, unless there be the complication of gonorrhœa or soft chancre, these diseases then taking their accustomed course, until the syphilitic infiltration begins at the point of inoculation.

Invasion.—The *primary* sore of acquired syphilis usually begins as a small pimple (papule), appearing upon an indurated base, about the fourth week after inoculation. This may slowly increase in size, for from two to four weeks, and then mildly ulcerate, over a small surface in the center. Or, the papule may begin to ulcerate at once, and assume the character of an indolent ulcer. In complicated cases, the primary sore is painless and insignificant, and causes little trouble, unless aggravated by heroic treatment, compressed beneath the prepuce, or irritated by chafing. Unless it be mixed with the virus of chancroid, it slowly passes through a protracted stage of mild ulceration for several weeks, without spreading, and then disappears, leaving, at its site, an indurated and reddened spot.

In from eight to fourteen days after the appearance of the primary sore, *painless enlargement* of the *lymphatic glands* begins, those traversed by lymphatics arising from the affected spot being first involved, the enlargement and induration gradually extending to the entire lymphatic system. The enlargement is not accompanied by active inflammatory symptoms, and suppuration never occurs, unless the syphilitic infection is complicated with secondary pus infection, an accident not liable to attend uncomplicated syphilis. When the genital organs are primarily affected, the glandular enlargement appears first in the inguinal region, in a week or two more the axillary glands become involved, and, in a week or two more, the cervical and occipital. These are now perceptible to the touch, enlarged, and hardened. Should the inoculation be made in a finger or hand, the lymphatic enlargement might be expected to appear first in the axilla of the corresponding side, in such instances the glands nearest the infected spot being first affected. Sometimes the lymphatics themselves are enlarged, feeling, under the finger, like hardened cords.

Secondary symptoms develop, from the sixth to the twelfth week after the appearance of the primary sore. These vary in constancy of order, though fever is often an early symptom. When this occurs, the temperature is not usually high, it varying from 101° to 103° F., though it occasionally reaches 104° or 105°. This is attended by headache, loss of appetite, muscular pain, insomnia, emaciation, and anæmia.

Pharyngitis is quite certain to come on early, and is especially severe if the patient has been recently mercurialized, the inflammation then extending to the *mouth*, and becoming ulcerative in character, both in the mouth and throat. The irritation is also liable to extend to the *larynx*, laryngeal cough and aphonia attending. Sometimes the ulceration is deep and extensive, rendering deglutition painful, and giving rise to much other local unpleasantness. Often the stomatitis involves the lips, stubborn, indurated *fissures* remaining here for months. Aggravated symptoms of this character are almost always due to the action of mercury, and seldom if ever appear, if this drug is avoided from the start.

About the eighth week, a *macular eruption* appears upon the abdomen, and spreads to other regions. It appears on the chest during the ninth week, on the shoulders the tenth, on the arms during the eleventh, on the forearms the twelfth, and on the hands, during the thirteenth. It is symmetrical, appearing on both halves of the body simultaneously, and is of a copper color. *Papules* appear about a month later. A row of these may be situated along the

margin of the forehead, constituting the *Corona Veneris*. *Pustules* may now appear, these being rarely seen however before the fourth month. They may be small and hard, feeling "shotty" under pressure, like the eruption of small-pox, or large, like the eruption of impetigo. They may ulcerate, and become covered by rupial crusts. Still later, a *squamous eruption* may appear, resembling psoriasis, and often termed "syphilitic psoriasis." This form appears most commonly on the palms of the hands, and soles of the feet.

It will thus be observed that the eruption of syphilis is *polymorphous*, none of the forms being distinctive of this particular disease, though the polymorphous character is peculiar to the development of syphilis. When the eruption invades mucous membranes, mucous patches, warts, and condylomata result.

Syphilitic eruptions are not usually painful, or sensitive. They are slow of development, and resisting to the influence of treatment, months being occupied in producing an impression on them with internal remedies, or local applications.

Alopœcia and *iritis* frequently occur, as symptoms of the secondary stage, especially in badly treated cases.

Tertiary syphilis, it is believed by many, is the result of mercurialization, rather than of the disease alone. The condition could not be brought about by mercury alone, and it is doubtful that the disease would reach such a stage without the pernicious influence of that drug. Eclectic physicians, of long-continued and wide experience in venereal diseases, aver that tertiary syphilis never follows, where mercurials are avoided and the patient has been properly treated from the start, with vegetable antisyphilitics.

The syphilides of this stage are unsymmetrical, and tend to excavate the tissues deeply. Round, deep ulcers occur upon the skin and mucous membranes. Sometimes the ulcerations upon the skin are tubercular and serpiginous, and these are always stubborn and chronic in character. Periosteal nodes appear along the shins, and these are accompanied by severe nocturnal (osteocopic) pains. Gummata, which undergo various degenerative changes at a later period, may develop in the skin, subcutaneous structures, or internal organs. When gummata develop in the viscera, they are liable to undergo fibrous transformation, with subsequent puckering and deformity, thus giving rise to serious obstruction of the function of the part.

The brain and cord, lungs, liver, digestive tract, circulatory system, kidneys and testes are all liable to the deposition of syphilitic gummata.

Gummata in the *brain* and *cord* form tumors, varying in size from that of a pea, to that of a walnut. They seem to develop from the

meninges, and are nearly always attached to the dura mater or pia mater. They may occur singly or in masses, and are most frequently developed in the cerebrum. They undergo a variety of changes, such as caseous, fibrous, or cystic degeneration. They do not occur as frequently in the cord as in the brain, though gummatous tumors have been found in all regions of this structure.

The presence of gummata in the brain and cord gives rise, in the early period of their presence, to *meningitis*, *arteritis*, and localized *foci of sclerosis*. Later, as the arteries become occluded, or local areas become debilitated by the meningeal inflammation, softening of the cerebral structures occurs. Or, cerebral hemorrhage may occur as a result of syphilitic arteritis, the weakened vessels giving way.

Tertiary lesions of this character usually come on years after the first appearance of the disease, though occasionally they appear within a few months. Psychical disturbances develop early in such cases, and the careful observer soon becomes convinced of structural cerebral disease. Delirium, either abrupt or preceded by headache, giddiness, etc., may appear, or there may be a gradual lapse into a condition of drowsiness and coma, while in other cases paretic dementia is the leading symptom. Convulsions may supervene, epileptic seizures sometimes alternating. Sometimes cerebral syphilis displays the symptoms of tumor of the brain. There is inflammation of the optic nerve, with headache, vomiting, convulsions, etc. Sometimes the early symptoms may be abrupt, resembling results of thrombosis or embolism, hemiplegia being the first indication of cerebral disturbance. In other cases, there may be loss of normal power of muscular coördination, the gait being staggering and unsteady, like that of a drunken person. When spinal syphilis occurs, the gummata are attached to the meninges, and imbedded in the substance of the cord. Meningeal inflammation may be provoked by their presence, and this may result in convulsions, or other reflex action. Sclerosis may develop from fibroid changes, locomotor ataxia being the result, or the various symptoms of compression of the cord may arise, from the presence of the morbid growth.

Syphilis of the lungs is common in the new-born subject of hereditary syphilis, and in acquired syphilis it occasionally occurs, coming on here after the second year. Gummata, varying in size from that of a pea to that of a marble, become deposited throughout the hepatic tissues. When these are numerous, the fibrous changes which occur cause such marked contraction that the organ becomes very much distorted and disfigured, so much so as to sometimes resemble a bunch of grapes. However, sometimes the gummata soften and liquefy instead of undergoing fibrous change, the diseased

organ becoming, where the morbid deposits have been numerous, soft and fluctuating. In some cases, Glisson's capsule may become thickened through the syphilitic influence, perihepatitis and increase of connective tissue giving rise to contraction and deformity. In many cases, the symptoms are those of hepatic cirrhosis. There are digestive disturbances, icteric symptoms, slightly marked, emaciation, and ascites. If the ascitic fluid be evacuated, and careful palpation be made over the right hypochondriac region, the marked irritability of the organ will be detected. In other cases, extensive amyloid degeneration of the liver may follow the deposition of the gummata, this involving the spleen and intestinal mucosa also, and there will be anæmia, albuminuria, and anasarca, or ascites.

In *syphilis of the digestive tract*, there may be syphilitic deposit in the œsophagus, stomach, small intestine, cæcum, or rectum. When gummata are deposited in the œsophagus, stricture is the result. Syphilitic ulceration of the stomach and intestines, as well as of the œsophagus, is rarely met. The common location of intestinal syphilis is the rectum, gummata being deposited in the submucosa, above the internal sphincter, the changes which follow giving rise to narrowing of the opening, and permanent stricture. These changes are gradual in their enchroachment, sometimes occupying years for the complete development of the rectal stricture.

Syphilis of the *circulatory* system may involve the heart or the arteries. A warty endocarditis occasionally occurs in syphilitic subjects, and gummata may develop upon the valves, giving rise to various secondary changes, such as fibrous or sclerotic. The *myocardium* may be the seat of gummatous growths also, these causing inflammatory action, and even rupture of the heart-wall.

The *arteries* may be occluded through a syphilitic arterio-sclerosis, or so weakened that aneurisms result. In obliterating endarteritis, there is proliferation of the subendothelial tissue, the hyperplasia occurring within the elastic tunics, and enchroaching upon the lumen, until the vessel is closed. This condition is not peculiar to syphilis, however, and not diagnostic of this disease, unless there are gummata in other parts, or there is a confirmatory history of syphilis. When nodular gummata develop in the adventitia, however, there can be no mistake. Globular tumors of varying size appear, especially upon the cerebral arteries, giving rise to inflammatory action in the surrounding tissues.

The *kidneys* are occasionally the seat of gummata, though these are not usually numerous. Cicatrices are found upon post-mortem examination, though there are no clinical symptoms which lead to their detection during life. Possibly in future time the further per-

fection of skiagraphy will enable the practitioner to determine their existence before death.

The *testicles* are frequently the seat of gummatous deposits, the growths occurring in indurated masses, in the substance of the organ, and not in the epididymis, as in tubercle. The gland becomes enlarged, but the swelling is painless, and does not tend to degenerative change. *Syphilitic orchitis* may arise independently of gummata, a fibroid degeneration, with increase of interstitial elements and gradual contraction of the organ, ensuing. This is a slow and painless process, involving one side particularly, it being recollected that tertiary lesions are not symmetrical, as in the case of secondary lesions.

Congenital Syphilis.

Pathology and Symptoms.—Congenital syphilis presents us with all the pathological conditions found in acquired syphilis, except that the primary lesions do not develop. If the disease appear while the child is yet in utero, it may be still-born (or survive a few months), with all the symptoms of bad cases of secondary and tertiary syphilis, combined. In still-born children, and even in those born alive, large areas of, and even an entire lung, may be affected with *white pneumonia of the fœtus*, a condition in which the affected part is consolidated, firm, heavy, and airless, presenting, upon section, a grayish-white appearance (white hepatization of Virchow); and miliary gummata are scattered through the structure. The alveolar walls are here thickened and infiltrated, and the cells are filled with desquamated and swollen epithelium.

Diffused *syphilitic infiltration of the liver* is often present. Though the organ preserves its form, it is large, hard, and unyielding to pressure. It is yellowish in appearance, resembling the color of sole-leather, and when cut, foci of infiltration are observable upon microscopical examination (miliary gummata) and connective tissue is found greatly increased in amount. Jaundice is frequently present, the icterus persisting until a fatal issue follows.

When the disease exists at birth, the child presents a wasted, wrinkled appearance, the abdomen is abnormally large, and there are cutaneous lesions, especially around the wrists and ankles (bullæ), and upon the hands and feet (pemphigus neonatorum). Snuffles are common with syphilitic babies, and fissures in the corners of the mouth and herpetic eruptions behind the ears, are nearly as common. Ulceration of the lips is often present. The bones are liable to be diseased, separation of the epiphyses usually being present in such cases.

A syphilitic child may be born healthy, and thrive, for a few weeks

before the syphilitic manifestations appear. Between the fourth and eighth weeks, however, irritation in the nasal passages becomes manifest, and persists, in spite of ordinary treatment for congestion of the Schneiderian mucous membrane. There are snuffles and mouth breathing, these being so urgent that the child may be unable to nurse. This syphilitic rhinitis becomes progressive, and a catarrhal discharge is soon established, varying from a seropus to blood. Ulceration, followed by necrosis of the nasal bones, may take place, unsightly depressions at the root of the nose often resulting. Continuing along the eustachian tube, the middle ear may be involved, destruction of important parts here terminating in permanent deafness. Simultaneously with the development of the snuffles, or soon after, *cutaneous eruptions* appear, first about the nates, in the form of irregular brown patches, or as eczematous or erythematous rashes. Sometimes papular syphilides appear here in the beginning. The mouth is involved early, the lips and tongue presenting ulcers and fissures, the child soon communicating the disease to the nipple of the wet-nurse, unless artificially nourished. The disease, in the form of infantile syphilis, is very infectious, not only the wet-nurse but other members of the household becoming contaminated, possibly through kissing, or the common use of towels or other toilet articles. The cuticular appendages, such as the hair and nails, are usually affected, the hair and eyebrows fall-out, and onychia developing. Laryngeal irritation becomes manifest in many cases, the voice being harsh and high pitched, the *cry* of the syphilitic child being thus peculiar. The glands are not generally enlarged, as in acquired syphilis, though where cutaneous lesions are severe and deep-seated, neighboring lymphatics may become affected. The liver and spleen are usually enlarged, and hemorrhages are not uncommon, these issuing from the gums and umbilicus, or into the subcutaneous tissue, forming hemorrhagic patches beneath the skin.

Syphilitic children usually perish before the period of infancy has passed, though they may survive, and continue to live through a protracted period of stunted growth. Childish peculiarities persist into years of adolescence. A syphilitic patient at twenty-one may not appear more than ten or twelve years old. If the child seems to have recovered during infancy, the disease is likely to reappear at puberty. Then he may present a wizened, wasted appearance, and a prematurely old look. The skin is sallow, and there are cranial peculiarities, which mark the presence of the disease. The peculiar appearance of such a patient is designated as "infantilism." The forehead projects, the frontal eminences are prominent, and the cra-

nium is asymmetrical. The Hutchinson teeth are present, these being characterized by a notched condition of the cutting edge of the middle incisors, which are peg-shaped—narrower at the extremities than at the gums. The bridge of the nose is sunken, and the tip is turned up (pug-nose). About the period of puberty, eye and ear affections are liable to develop. Of eye affections, keratitis and iritis are most common. The keratitis is interstitial, the cornea presenting a steamy appearance, sometimes one and sometimes both being affected. After a time, the cloudiness may clear up, though spots or specks of opacity may remain permanently. While a variety of ear affections may be due to syphilis, a peculiar kind may develop about puberty, in which deafness comes on rapidly and remains permanently, in spite of treatment, and in which there are no obvious local lesions, the pathological changes probably affecting the labyrinth. The bones may be involved, both early and late. Some of the marked cases of chronic gummatous periostitis may be mistaken for rickets.

Synovitis, enlargement of the spleen, and gummatous deposits in the liver, kidneys, and brain, may all occur as late manifestations of hereditary syphilis, as well as of the early stages.

General Diagnosis.—Syphilis may exist in obscure form, and be the underlying factor in the obstinacy of many cases which would otherwise improve rapidly under medication. A proper treatment, as well as prognosis, depends, then, upon the ability of the practitioner to recognize and provide for them. There is a disposition on the part of many patients to conceal the fact, when they have been affected by acquired syphilis, and the physician must be prepared for this, and draw his conclusions accordingly. As few are acquainted with all the symptoms liable to follow, however, careful questioning will enable the physician to come very near the truth, however well the patient may attempt to guard it. The history of throat and skin lesions, loss of hair, emaciation, etc., occurring as associated symptoms, are very good evidence, when attending conditions already suggest the disease. In primary syphilis, the patient may contract gonorrhœa coincidentally, and the chancre be concealed within the urethra, along the fossa navicularis; but the presence of enlargement and induration in the inguinal region, and the development of mucous and cutaneous lesions later, with fever and loss of strength, will convey intelligence of the specific character of the disease. In advanced cases, nodes on the shins or other parts of the skeleton, old scars, and more or less thickening of the lymphatic glands, especially in the inguinal and occipital regions, are confirmatory of a suspicion of the presence of syphilis. In congenital syph-

ilis, there is little danger of mistaking the disease. The early appearance of snuffles, in conjunction with cutaneous and mucous lesions, can hardly be mistaken for any other affection. The peculiar developments at puberty, already described, will be confirmatory testimony, as the case progresses.

Prognosis.—In these days, the old virulence of syphilis seems to have become nearly exhausted. With rational treatment, few cases of acquired syphilis will result seriously, or even develop very unpleasant secondary symptoms, while tertiary manifestations may generally be entirely avoided. Unfortunately, many cases of this disease fall into the hands of old school physicians, who adhere to the stupidly pernicious practice of administering mercury during the early stages. The result is, that the following lesions are more severe, and much more difficult to control. We can promise much more to a patient who has avoided mercury throughout, than one who has been subjected to the action of that drug for several weeks' time in the start. It is possible that mercury may suppress the cutaneous lesions somewhat at first, but if these are allowed to come out, while the use of proper vegetable antisyphilitics is made, they will usually disappear permanently, within a brief period. Mercury invariably aggravates the mucous lesions about the mouth and throat, these becoming increased and prolonged, as mercurial treatment is persisted in.

Congenital syphilis is less amenble to treatment than the acquired form, though it is possible that early treatment, through the maternal circulation, might avert many of its evils.

Treatment.—*Preventive treatment* is always to be considered. A syphilitic patient should be warned to avoid the common use of toilet articles, drinking cups, pipes, etc., with uncontaminated persons. A syphilitic child should be reared on the bottle, unless its own mother nurse it, and precautions should be observed as to the use of towels, combs, drinking-cups, etc., and other children, as well as adults, should avoid kissing and fondling it. The person who attends to washing its clothing should see that there are no abrasions on her hands, through which the virus may find entrance to the circulation. With an intelligent idea of the nature of the disease, suggestions as to proper care to guard against infection will naturally arise, in the mind of every thinking person.

The *medicinal treatment* of acquired syphilis is simple, and, at the same time, effective. *Berberis aquifolium* is as near a specific for syphilis as we can hope for, in any case. But, as syphilis is a disease of slow development and chronic course, we must not expect a few weeks' treatment to eradicate it. Many patients improve so rap-

idly upon it that they finally, in a few weeks, consider themselves cured, and then abandon treatment, to their ultimate sorrow; but when continued for months and years, nothing could be more satisfactory than the results thus obtained. Syphilitics, in the wasting stage of the disease, improve in appetite and flesh under this remedy, debility disappears, and former vigor is soon restored. Periosteal pains subside under its influence, and gummata are averted, though it possesses no power to discuss them, when once formed. But the ulceration which follows their breaking up in the skin and mucous membranes becomes less stubborn, and usually heals within reasonable time. It removes the cutaneous eruptions of secondary syphilis in a few weeks, and assists in healing the patches in the mouth and throat.

Corydalis formosa is a remedy which rivals berberis aquifolium. To avert gummatous periostitis and prevent the formation of nodes, it is probably without a rival. It combines well with berberis, and I am in the habit of prescribing as follows, in most cases of syphilis, for constitutional purposes: ℞ Fluid extract berberis aquifolium ʒi, specific corydalis ʒss, alcohol ʒii, aqua, ad. q. s., Oi. Sig., Take a tablespoonful four times daily. This combination will answer every purpose, where syphilitic cases have not previously been subjected to the baneful effects of mercury. When this drug has been used, however, tertiary lesions may be stubborn, and demand the employment of *iodide of potassium*, to hasten the liquefaction of syphilides, when breaking down begins in the skin and mucous membranes. This may then be given in tolerably large doses (gr. x) for a few weeks, to be temporarily discontinued, until the patient has time to recuperate from the resulting debility, upon berberis. It is well, in such cases, to alternate the berberis with the potassic iodide, as well as follow with its temporary administration.

Stubborn cutaneous eruptions, following upon the administration of mercurials, may sometimes yield to large doses (gtt. x) of *Donovan's solution*, repeated three or four times a day. It may be suggested that this combination contains mercury, an objection which can hardly be raised to its use when the system has already been thoroughly poisoned with the drug. Sometimes stubborn cutaneous lesions, such as palmar psoriasis, can be cured by the persistent local use of Webster's *compound sulphur ointment*: ℞ Lanolin lb. i, ol. tar ʒi, sp. m. veratrum ʒi; thicken with powd. sulphur. This should be applied morning and evening, for a year, constantly.

The primary sore of syphilis needs little attention, unless it be developed where it is subjected to pressure or chafing. Twenty drops of nitric acid, diluted in four ounces of water, constitute a

cleansing wash, which tends to heal the abrasion and prevent the growth of condylomata. It should be applied four or five times daily. When the chancre is concealed beneath a constricted prepuce (phymosis), aggravation is liable to result, and an irritable, ragged ulcer may arise from the pressure. In such a case, the prepuce should be slit freely, so that all compression may be avoided; the lotion of dilute nitric acid will now suffice to readily heal the abrasion.

Echinacea has been presented, by modern Eclectic physicians, as a remedy for syphilis. My experience has been confined to its use where stubborn pharyngeal ulcers have manifested an irritable, sensitive condition. Here, echinacea, both locally and constitutionally, produces satisfactory results. Sometimes the local use of galvanism is useful to assist in healing painful and stubborn pharyngeal and palatal ulcers, two or three milliamperes being applied to the spot with the negative pole, and repeated every other day.

As constitutional remedies for this disease, additional to what have already been mentioned, may be named *chaulmoogra oil*, which may be administered in ethereal solution, or in capsules.

Stillingia sylvatica, in the form of a green plant tincture, combined with iodide of potassium, is especially recommended in tertiary syphilis. *Chloride of gold*, in tertiary syphilis of the bones, in second decimal trituration, is worthy of trial.

Professors Goss, of Georgia, and J. W. Hamilton, of California, are enthusiastic admirers of echinacea, administered in from thirty- to sixty-drop doses of a saturated tincture of the fresh root.

XXVI. LEPROSY.

Synonyms.—Lepra; Elephantisis Græcorum; Leontiasis.

Definition.—A chronic infectious disease, caused by the bacillus lepræ, characterized by tuberculous growths in the skin and mucous membranes, and areas of anæsthesia and destructive ulceration, corresponding to the distribution of nerves which become affected by the development of the bacilli in their structures.

Etiology.—Leprosy is a disease of the earliest antiquity—known to the earliest writers. It prevails along the shores of the Mediterranean sea extensively, though since the middle ages it once nearly disappeared from Europe, except in Norway and the Orient. On the Pacific Coast, the disease is occasionally found among Caucasians, and the Chinese are frequently affected. The Sandwich Islands are notoriously affected, there being over a thousand lepers at the settlement of Molokai. At Tracadie, N. S., is a lazaretto, in

which are confined about a score of lepers, the disease having been introduced by emigrants from Normandy, during the latter part of the seventeenth century. The number of persons affected is diminishing, the settlement having formerly contained over forty members. It is most liable to spread in hot climates, the West Indies, the Gulf States, and Mexico, being homes of quite a large number of lepers. All ages and all classes may be affected.

The exact method of transmission is not positively known, though it is generally believed that the disease may be propagated by sexual congress. It certainly is not very contagious, unless there be special exposure, for healthy persons may be about lepers for years, and remain uncontaminated. Osler states that not one of the Sisters of Charity, who for forty years have nursed the lepers of Tracadie, have contracted the disease. It is believed to be hereditary.

Dr. Morrell Mackenzie, in an article written a short time before his death, declared that leprosy is alarmingly on the increase, all over the world. He asserted that in Spain and Portugal, as well as in other parts of Southern Europe, the disease is rapidly gaining ground, the fact being due, according to his belief, to the lax provisions of the proper authorities for isolating those affected. He was a firm believer in the contagiousness of the affection, and deplored the custom of so many of regarding it as non-contagious, since such belief led to lack of proper isolation of those affected. Jonathan Hutchinson and others ascribe the complaint to diet, Hutchinson believing that a fish diet tends to its production. A large contingent scout such a proposition, however, asserting that there are many facts in history which contradict such a statement.

The *bacillus leprœ* resembles the bacillus tuberculosis in many respects, though, as has already been pointed out (in the Introduction), there is a distinction. It can be found in the tuberculous structure of leprosy, in large numbers, though it does not propagate in inoculation tests upon animals, thus differing decidedly, in one respect, from the bacillus tuberculosis.

Pathology.—Like tubercle, the tuberculous growths of leprosy are due to granulomatous infiltration, from the irritating influence of the bacilli upon the embryonal cells of connective tissue. The growth involves the skin later, and grows outward, tuberous projections forming over circumscribed areas, between which are ulceration or cicatrization. These give rise to disfiguration of the surface, and, when the face is involved, remarkable distortion of the features may result. Sometimes deep ulceration may further disfigure the part, amputation of the fingers and toes thus occasionally resulting. When the bacilli develop in the substance of the nerve-fibers, there

is more or less destruction of their functions, and peripheral neuritis results, with localized areas of anæsthesia, and trophic changes in the skin. The bacilli are found in great numbers among and within the cells of the tuberculous growths, and among and within the affected nerve-fibers.

Symptoms.—Two *clinical forms* are described, viz., tubercular leprosy, and anæsthetic leprosy:

The first appearance of *tubercular leprosy* may be that of sharply defined spots upon the skin, resembling erythema or psoriasis. Sensibility may here be exalted, at first, and, after a time, the spots may become pigmented. Anæsthesia gradually develops, and there is either a gradual outgrowth of tuberculous nodules over the surface, or the spots gradually fade out, becoming perfectly white (lepra alba). The mucous membranes become involved gradually, the voice growing hoarse and husky, and finally being entirely lost, from involvement of the laryngeal mucous membrane; and death may result from laryngeal complications, giving rise, later, to pneumonia, of chronic form. The eyebrows and eyelashes, as well as the other hairs of the face, fall out; tuberculous growths may form upon the conjunctivæ, resulting in blindness from leprous keratitis, and the face may become frightfully thickened and distorted, from the cutaneous tubercular outgrowths. The most common locations of the growths are the face, breast, scrotum, and penis.

Anæsthetic leprosy differs materially in its characteristics from the tuberculous form, though due to the same cause. Here the disease is largely confined to nerve-trunks, while its outward manifestations are exhibited upon the surface, in peripheral results of disturbed or arrested sensory or trophic functions. The earliest symptoms are *pains* in the limbs, and areas of *hyperæsthesia*, *anæsthesia*, or *numbness*, upon the surface. Maculæ or pigment-spots may appear upon the trunk and extremities, here to persist for a time and afterward disappear, leaving localized areas of anæsthesia. Bullæ upon the surface may denote trophic disturbances, these appearing, in some cases, quite early. Enlargement and nodulation of superficial nerve-trunks may be felt, after the disease has progressed for a time, and the trophic disturbances become more and more marked, as the disease progresses. Pemphigus-like bullæ form and break, leaving deep and destructive ulcers; contractures and necroses of the fingers and toes, and other destructive changes, follow. The changes are persistent and gradual.

Diagnosis.—The macular spots, with hyperæsthesia and subsequent anæsthesia, will suggest the character of the disease early. Later, the development of tuberculous growths on the face and other

parts, with attending symptoms, could hardly be mistaken; and the manifestations of anæsthetic leprosy are about as positive.

Prognosis.—As there has not yet been discovereda cure for leprosy, and the tendency is continually, though slowly, toward a worse condition, the prognosis, as to a cure, must necessarily be unfavorable, though life may not be materially shortened by the disease.

Treatment.—The treatment of leprosy is not liable to bring laurels to the attending practitioner. There are no remedies which produce striking results, and we cannot expect to do more than lessen the rapidity of its progress. Lepers hardly continue treatment long enough to give any remedy a fair trial, and few are appreciably benefited with drugs. *Chaulmoogra oil* and *gurjun oil* have been recommended. *Berberis aquifolium* should be thought of favorably. In any case, treatment should be persisted in for a long time—many months—if improvement is to be expected.

The protection of the uncontaminated public is more important than the cure of a few individual cases. Lepers should be isolated and confined, so that the disease may not spread. This is a matter in which law-makers in all civilized countries should act together, that proper lazarrettos may be instituted, where such subjects can be provided for, and restricted from intercourse with the world at large.

XXVII. GLANDERS.

Synonym.—Farcy.

Definition.—A specific, infectious disease of horses, communicable to man, characterized by the formation of nodules of granulomatous tissue, occurring chiefly in the nares (glanders) and beneath the skin (farcy).

Etiology.—The cause of this disease is a short, non-motile bacillus, resembling the tubercle bacillus, which enters the body through an abraded surface, either of the skin or the nasal mucous membrane, the disease usually being contracted from affected horses, though it may be communicated from man to man, washer-women having been inoculated from the clothing of those affected.

Pathology.—The disease consists in the formation of granulomatous tumors, of low vitality, which tend to early breaking down. They are composed of epithelioid and lymphoid cells, among which are found the bacilli—the irritating elements which provoke the morbid growth. These nodules manifest a tendency to undergo rapid destructive changes, which result in ulceration of the mucous membrane and deeper structures of the nose, in glanders, and in abscesses beneath the skin, in farcy. Internal organs sometimes become

involved, and the characteristic nodules are then found in their structure.

Symptoms.—This disease may occur in either the acute or chronic form, both these occurring in the nasal and subcutaneous varieties.

When the *nose* is involved, an acute attack is inaugurated by general febrile disturbance, with redness, swelling and lymphangitis at the point of inoculation, nodules soon forming about the vicinity, which break down a few days later, melting away in a profuse, mucopurulent discharge. The cervical lymphatics soon become swollen, hardened, and painful. Associated with these symptoms is a cutaneous eruption, first papular, then pustular, which appears on the face and about the joints, resembling, in general appearance, the eruption of variola. Severe constitutional symptoms attend the local manifestations. There are fever, rapid prostration, and typhoid symptoms, the disease terminating fatally in from eight to ten days. When the chronic form occurs, the symptoms are at first those of a severe coryza. There is ulceration of the nasal mucous membrane, with laryngeal irritation and ulceration, which may linger for months, recovery finally taking place in some cases, though a fatal termination usually follows.

When the *skin* is involved in acute farcy, there is severe phlegmonous inflammation at the point of inoculation, with rapidly spreading swelling, the lymphatics becoming involved, and nodules (farcy buds) forming along their course. These soon reach a stage of suppuration, and abscesses form in the vicinity. Pain and swelling occur about the joints, though the eruption observed in the nasal form is rarely met. Severe constitutional symptoms, similar to those of septicæmia, rapidly develop, and a fatal termination is almost inevitable, within twelve or fifteen days. Chronic farcy is more gradual in its inception, and it is characterized by the presence of localized tumors, usually in the extremities. These break down into abscesses and form deep ulcers, without much constitutional disturbance. In chronic farcy, the lymphatics are not usually involved, and the disease may continue for months or years, recovery occasionally resulting finally, though pyæmia and death follow more frequently. Sometimes acute glanders may result from auto-inoculation.

Diagnosis.—The diagnosis will not be difficult, the severity of nasal glanders distinguishing it from all other forms of nasal trouble, unless the chronic form occur. Here, it may be necessary to submit some of the discharge to a bacteriologist, for culture and other inspection. The history of the case, the "farcy buds," and early sub-

cutaneous abscesses of acute farcy, can hardly be mistaken for any other disease, especially when the history of the case will usually afford evidence of exposure to infection from a diseased horse, at a recent date.

Prognosis.—The prospects of recovery from acute glanders or farcy are exceedingly doubtful, and the prognosis must be almost invariably unfavorable. Recovery from chronic glanders and farcy sometimes occurs, and there may be some hope offered, though even here there is little prospect of a favorable termination. It is said, however, that the noted French veterinary surgeon, Bouley, recovered from an attack of chronic farcy.

Treatment.—*Prophylaxis* is the important part of treatment, because restorative treatment is not attended by very promising results. If the point of inoculation can be discovered early, it is advised to excise, or destroy it with caustics. Farcy buds should be opened early, and thoroughly drained, with local antisepsis. Internally, we may derive some benefit from the persistent use of echinacea or berberis aquifolium, preference being given to echinacea in acute cases, and to berberis aquifolium in chronic ones. In acute attacks, either of nasal glanders or farcy, little hope can be offered, though this need not deter us from trying the best Eclectic remedies in our possession.

XXVIII. ACTINOMYCOSIS.

Synonyms.—Big-jaw; Lumpy-jaw.

Definition.—A specific infectious disease of cattle, pigs, horses, and other animals, communicable to man, caused by the ray fungus (actinomyces).

Etiology.—The *actinomyces* is a fungus, consisting of microscopic threads, radiating from a common center, bearing, on their ends, bulbous or club-like terminations. Infection probably occurs from feeding, as the tissues about the jaws are usually affected first, though the disease may originate in the intestines, lungs, brain, or skin. It is believed that barley or rye may contain the fungus, and be a source of the disease among cattle, if not men. Doubtless diseased flesh also conveys it to man. It is asserted that the fungus may gain entrance through abrasions in the skin and mucous membranes, and through cavities in decayed teeth. The disease is rare.

ACTINOMYCES.

Pathology.—The pathological change consists in a conversion of mature connective tissue into a granulomatous mass, composed of

round and epithelioid cells, with occasional giant cells, the growth, in its early condition, resembling that of ordinary tubercle. After a time, however, there begins a rapid growth of the tumors, owing to active proliferation of the connective tissue in the neighborhood, the morbid condition then much resembling sarcoma, in its general appearance. Suppuration begins later, and the pus contains yellow particles, visible to the naked eye. The growth now becomes burrowed with fistulous sinuses and scattering abscesses. Chronic inflammation of the surrounding tissues attends, though the lymphatics do not become involved. The later course of the disease resembles that of a malignant tumor.

Symptoms.—These vary, to correspond with the location of the morbid growth. If the *face* is primarily affected, an irregular, nodulated swelling will involve the cheeks, jaws, temples, tongue, or some contiguous part, with slow and painless enlargement, at first, though it will take on a rapid growth later. When suppuration begins, and irregular fever attends, the condition resembles that of chronic pyæmia. Septic symptoms may be prominent, and the symptoms may simulate those of typhoid fever. When the lungs are involved, *cough* will be a prominent feature, and the disease will run a course similar to that of some cases of pulmonary tuberculosis, or fœtid bronchitis. When the *skin* is involved, nodular excrescences appear upon the surface, which ultimately ulcerate, and pass through a protracted period of suppuration, the ulcers stubbornly remaining for years, thus bearing a resemblance to tuberculosis of the skin. When the *brain* is the part affected, symptoms of cerebral tumor are manifest. Epileptic symptoms, unsteadiness of gait, and mental disturbance, in the beginning, with delirium and coma, later on, are liable to appear. When the *intestines* are involved, gastro-intestinal disturbance will be prominent.

The disease may involve parts distant from the face, secondarily, there then being a complication of the disturbance about the face with the various visceral troubles.

Diagnosis.—The presence of the actinomyces in the pus will be the distinctive diagnostic feature, though when the location of the affection is in its usual part, about the face, the general picture and peculiar course will be highly suggestive. The yellow particles in the pus, often visible to the naked eye, demonstrated, upon microscopical examination, to be actinomyces, will settle the question.

Prognosis.—Wherever the disease is located, there is always liability of secondary infection. Its course is likely to resemble, clinically, that of sarcoma, and it almost invariably goes on to a fatal termination. However, when the disease is restricted to the skin,

or is located so superficially as to permit of surgical interference without involving vital parts, recovery may follow early treatment.

Treatment.—The treatment is principally surgical. If the disease be located where it can be exposed, the surgeon's knife should be called into service, to eradicate every vestige of the morbid growth. When the case progresses to suppuration, the treatment pursued in pyæmia is all that can be offered.

XXIX. INFECTIOUS DISEASES OF DOUBTFUL NATURE.

Simple Continued Fever.

Synonyms.—Febricula; Synocha; Synochoid.

Definition.—An infectious fever, usually of short duration and favorable prognosis.

Description and Etiology.—In non-malarious districts, during the absence of epidemics or endemics, febrile affections occur, which arise from colds, retained secretions, errors in diet, excessive mental or physical effort, exposures to the sun, or other accidental cause, outside of any known specific infection, which may be classed under this name. There is no regular or stated course of continuance, in this class of fevers, the gravity of the cause, the constitutional resistance of the patient, or the treatment, determining the period of duration. In some cases, the fever will terminate in a day or two, while in others, it may continue for from ten days to three weeks.

Sometimes the system is in such a predisposing condition that the fever assumes quite a serious aspect, and takes on a high grade of temperature and pulse-rate, the maximum temperature reaching as high as 106° for several days, and the pulse running at a rapid rate, full and bounding. Such patients possess powerful reactive constitutions, and the course of the fever is actively inflammatory in its characteristics, tending to inflammation of the lungs or brain, if it does not terminate within the first week. This character of febrile manifestation has been described as a separate form, under the name, "synochal fever." After this time, typhoid symptoms gradually appear, and delirium and blood depravation, as manifested by the condition of the tongue, develop. Or, typhoid symptoms may appear early in the course of the disease, within the first two or three days, and the temperature may run a course much like that of typhoid fever, during the fastigium, the prostration, nervous symptoms, and blood depravation simulating that disease very much, though the fever may terminate within two weeks, usually, under

rational treatment. This form has been described by some writers under the term, "synochoid," or "common continued fever."

Symptoms.—The disease usually begins with an abrupt rise in temperature, the stage of invasion only occupying the first day, or a few hours. The temperature may rise as high as 102°, 103°, 104°, 105°, or 106°, during the evening, but, if the fever lasts over the second day, there is a morning remission of one or two degrees, each day. Where the temperature is very high, the form known as *synocha* may develop, and the disease run for a week, the pulse being full and bounding, respiration hurried, and the patient restless and wakeful. There is headache during this stage, the eyes are bright, the urine is scanty, and perspiration is arrested, the skin being hot and dry, the bowels constipated, and the appetite absent. The patient is usually more restless and uncomfortable during the after part of the day and early part of the night, morning hours being attended by subsidence of the more aggravated symptoms. Continuing in this way for five or six days, favorable cases terminate suddenly, by rapid lysis, or crisis; secretion becomes established, the urine flows freely, the skin becomes moist and cool, the pulse normal, and respiration easy and natural, the headache subsides, and the appetite returns.

If the fever does not abate at this time, and, also, if there has been considerable of a period of incubation, the symptoms from the start may assume a typhoid character. This constitutes the form known as *synochoid*, or *common continued fever*. When so from the beginning, there is usually a marked chill, following several days of depression. Though not so severe as an ague, the patient will complain of coldness of the extremities, and of chilly sensations, creeping over the body. These are soon alternated with flushes of heat, until febrile action is well established. Now we begin to note the typhoid symptoms. The tongue is soon coated; the coating may be pasty white or it may be yellowish, or there may be a tendency to an irritable condition of the stomach, as indicated by the elongated tongue, with reddened tip and edges. Whatever the condition of the tongue in the commencement, it is liable, in the later stages, to be either clean and slick, with dark red mucous membrane, or the coating to become brown and dirty. In bad cases, there may be sordes on the teeth and lips. The pulse is now small and feeble, and the patient is liable to develop considerable disturbance of the cerebral centers, as manifested by dreamy delirium, or coma-vigil. While this condition resembles that of typhoid fever, in many respects, there is usually absence of diarrhœa, tympanites, and other abdominal symptoms characteristic of true typhoid. Pulmonary

complication often attends protracted cases of this fever, and, occasionally, serious cerebral congestion.

Diagnosis.—The sudden onset and early decline of the fever, without complication, will enable one to readily diagnose the simpler cases. Where typhoid symptoms appear, and the disease becomes protracted, there will be an absence of the serious abdominal symptoms that characterize true typhoid. The rash of typhoid fever will also assist in clearing up a doubtful diagnosis. It should be recollected that typhoid fever occurs as an epidemic or endemic, and that sporadic cases can seldom be supposed to exist, while the opposite is true of this form. Where it is necessary to render a diagnosis in obscure or doubtful cases, microscopical inspection of the supply of drinking water and milk, and the evacuations, may assist in clearing up the obscurity.

Prognosis.—There is little danger of a fatal termination, even in the most aggravated form, if judicious management be observed. Though the synochoid form may present some aggravated features, proper treatment will usually correct them in good season, and a favorable termination in all cases, except those of great debility or extreme age, ensues.

Treatment.—Abbreviated cases of febricula require little treatment, more than that which will render the patient less uncomfortable. Small doses of *aconite* and *gelsemium* may be employed to lessen the height of the fever, and a full dose of *bromo-seltzer* may be administered, if there is severe headache. Cooling lotions may be applied to the head, and mildly acid drinks administered, until the attack passes off.

The *synochal* form will demand the use of *jaborandi*, as follows: ℞ Sp. m. jaborandi ʒiii, water ʒiv. M., and give a teaspoonful every hour, until the fever declines. When the pulse is bounding, with other *gelsemium* indications, that agent may be preferred. An alkaline sponge bath, administered every day, or a cold abdominal wet pack, will assist in reducing the fever, with safety to the patient.

The *synochoid* form may be treated as a case of typhoid fever. Sometimes the indication for some special antiseptic will be pronounced, and the important part of the treatment will consist in supplying this demand. For more definite instruction here the reader is referred to the general treatment of fevers, in the Introduction.

Weil's Disease.

Synonyms.—Acute Infectious Jaundice; Bilious Typhoid of Griesinger.

Definition.—An infectious disease, characterized by marked

jaundice, high fever, severe pains in the extremities and back, and albuminuria, with termination, in from ten to twelve days, by lysis.

Etiology.—The exciting factor of this disease is unknown. It is most liable to occur in hot weather, among males between twenty and forty years of age, and is especially liable to affect butchers, these facts suggesting that exposure to putrefactive exhalations from animal and vegetable decomposition may exert a causal influence. Mild epidemics may occur.

Pathology.—Little is known of the morbid anatomy of this disease, as it seldom proves fatal. The symptoms suggest obstructive jaundice and renal irritation. There is evidently splenic engorgement, detected by palpation during the course of the disease. The kidneys are congested, with acute parenchymatous degeneration of the histological elements.

Symptoms.—The onset is abrupt, there being a chill or succession of rigors followed by high fever, the temperature rapidly rising to 104° or 105°, and the pulse to 100 or 110 per minute. The fever is remittent, and remains high for about the first week, when it declines by lysis, terminating about the tenth or twelfth day. The jaundice appears early, often on the second day, the icteric hue being deep yellow, in the skin and conjunctivæ, the tongue being loaded with a yellowish fur. There is nausea, disgust for food, and sometimes vomiting, in the beginning, and a diarrhœa of clay-colored stools is liable to appear later. Severe headache, thirst, backache, and pains in the extremities mark the exacerbations, while the remissions are attended by little amelioration. Bile, along with albumin, is present in the urine. As the disease progresses, it becomes less active, and the patient is finally prostrated, and may manifest more or less marked typhoid symptoms.

In about one-fourth of the cases, a relapse occurs six or seven days after the return to normal, the temperature again rising, as in relapsing fever. The relapse is mild, however, and only lasts five or six days. Convalescence is slow and tedious, sometimes occupying two or three months.

Diagnosis.—The symptoms of jaundice occur too early for that which sometimes arises in relapsing fever, and the history of the case will usually distinguish between this disease and that. In typhus fever the jaundice also occurs later, while the rash of that disease, about the sixth or seventh day, is characteristic. The history of the case, and the markedly epidemic character of typhus, will assist in distinguishing it.

Prognosis.—This is almost universally favorable, few cases resulting fatally.

Treatment.—The treatment will be adapted to each particular case. To control the fever and assist in eliminating the morbid elements from the system, the properly selected sedative should be administered every hour, during the first week, at least. Where there is nausea with indications of gastric irritability, *aconite* and *rhus* should be given: ℞ Green plant tincture of rhus tox. gtt xv, Lloyd's aconite gtt. v–vii, water ʒiv. M., and order a teaspoonful every hour. Or, where there is less tendency to nausea, more pronounced sedatives may be employed. ℞ Specific jaborandi ʒii–iii, water ʒiv. M., and order a teaspoonful every hour. Determination of blood to the brain will call for *gelsemium*, and capillary congestion for *belladonna*. In connection with the sedative, or alternated with it, the following should be administered every two hours, in teaspoonful doses: ℞ Sp. m. polymnia ʒi, sp. m. chionanthus ʒss, sp. m. chelidonium ʒi, water ʒiv. If the tongue is heavily loaded with a dirty, yellowish-white coating, *sulphite of sodium* should be given, in one-grain doses, every three or four hours, until the coating has disappeared. A bland and nutritious diet should be allowed during convalescence, and some appropriate bitter tonic should stimulate the recuperative functions, attention being especially paid to the demands of a malarious district.

Milk Sickness.

Definition.—A disease supposed to be communicated to man from eating the flesh or drinking the milk of cattle affected by what is commonly known as "trembles."

Etiology.—This disease formerly prevailed among the early settlers of the states bordering on the western slope of the Alleghany Mountains. Cattle and sheep were subject to a peculiar nervous affection called trembles, characterized by refusal of food, injection of the eyes, and staggering gait, with trembling of the muscles, and, finally, death in convulsions. The cause of this disease has been supposed to be some form of plant-food taken with the wild herbage, the disease having gradually disappeared, as clearing up of the forests, and cultivated fields, have been the order. In some sections of North Carolina, it still prevails. When milchers are affected, it is said that they may not manifest the disease unless overdriven, the poison lurking in the milk and proving fatal to those consuming it. Sheep, as well as domestic cattle, may be affected, their flesh, as well as that of beeves, proving poisonous when eaten. It is said that an ounce of butter or cheese from an affected cow, or four ounces of beef, raw or cooked, three times daily, will prove

fatal to a dog, within six days. Nothing definite is known respecting the specific principle of the disease.

Pathology.—Little has been recorded of the pathology of this affection, as few scientific investigations by autopsies have been made. Doubtless the principal lesions will be found in the alimentary canal and cerebro-spinal centers.

Symptoms.—The symptoms of the disease in man are characterized by two or three days of prodromes, such as restlessness and gastric discomfort, followed by acute pain in the stomach, with nausea and vomiting, thirst, and fever, which rapidly passes into typhoid symptoms, the tongue becoming swollen and tremulous, the breath fœtid, the bowels constipated, and the urinary secretion more or less diminished. There is great restlessness and irritability at first, but this may give way to coma and convulsions. Death may occur in three or four days, or the disease may run three or four weeks.

Diagnosis.—The rapid onset of the disease, with the violent gastro-intestinal irritation and nervous symptoms, will suggest its presence, in sections where it is likely to prevail.

Prognosis.—The disease has fortunately become rare, as it is nearly always fatal in its results. The profound poisoning seems to defy the best treatment that has yet been tried.

Treatment.—Opiates should be avoided, and remedies administered to control the vomiting. *Bismuth*, and *aconite* and *rhus tox.* may be tried, for this purpose. When the vomiting has been arrested, *echinacea* should be administered freely. *Passiflora* and *lachesis* may also be thought of.

Malta Fever.

Synonyms.—Mediterranean Fever; Neapolitan Fever; Rock Fever.

Definition.—A febrile disease, which prevails at the Island of Malta, Naples, and other points about the Mediterranean Sea, characterized by an initiatory attack of mild febrile action of about a week's length, followed by a remission of two or three days, with a prolonged relapse of increased severity and persistent duration, during which gastro-intestinal, pulmonary, cardiac, and arthritic disturbances are liable to develop.

Etiology.—The nature of this disease is yet in dispute, it being generally denied that it is due to malaria. Some have asserted that it is typho-malarial fever, there being marked febrile exacerbations and remissions; but it does not yield to quinine, and does not behave like ordinary malarial fever. An examination of the blood will fully

settle the question. Some have contended that it is typhoid fever, but there is absence of the characteristic lesions of that disease. Rheumatic symptoms are sometimes present, and, catarrhal manifestations being prominent, it may be due to atmospheric influences. Sewer-gas has been suggested as a possible causal factor, though it is doubtful if any one has yet named the proper one.

Pathology.—There is irritation of the gastro-intestinal and pulmonary mucous membranes, with enlargement and congestion of the spleen, endocarditis, and effusion into the joints and other serous cavities.

Symptoms.—In the commencement, the symptoms may resemble those of mild quotidian ague, though usually the invasion is more insidious, and the patient may be unable to name the day upon which his illness began. Anorexia, lassitude, drowsiness, and slight headache are the first symptoms here, and these gradually advance, until there is nausea, vomiting, and diarrhœa, a few days later. Febrile symptoms alternated with chilliness now appear, and the severity of the symptoms increases day by day. Severe frontal headache has developed by this time, and the patient is sleepless, restless, nauseated and thirsty, constantly. In about a week, in mild cases, these symptoms abate, and the patient supposes himself convalescent, and goes about his duties. In two or three days however the old symptoms return, with increased severity. The nausea and vomiting are more aggravated, and there is active diarrhœa, this sometimes amounting to dysentery, with severe tenesmus and the evacuation of muco-sanguineous stools. Again, there may be symptoms of pneumonia, with cough and rusty sputum. In other cases, the prominent symptom may be that of excruciating pain in the back or one of the extremities, which is so severe as to prevent motion. There is steady loss of flesh, anæmia comes on with loss of hair, enlargement of the spleen and liver, and the patient slowly drags through a protracted convalescence, with extreme debility. The febrile symptoms are marked by periodicity, with evening exacerbations and morning remissions.

Treatment.—The treatment should be in accordance with the suggestions in the general treatment of fevers given in the Introduction. Hygienic precautions should be especially observed.

Miliary Fever.

This disease, which is otherwise termed "sweating sickness," prevailed in various parts of Europe and England during the fifteenth and sixteenth centuries, but it has been confined, during later times, to certain districts in France and Italy. When it occurs, large

numbers of persons are attacked at once, the disease spreading rapidly, like influenza. The disease is characterized by fever, profuse perspiration, and an erythematous eruption surmounted by a crop of miliary vesicles. Severe cases are attended by determination of blood to the brain and active delirium at first, with prostration and coma later on. Death sometimes occurs in a few hours, the outset of epidemics often being attended by a high death-rate.

Mountain Fever.

A severe form of fever prevails in elevated regions of the Rocky Mountains, to which this term is applied. Two varieties are described, one a continued, and the other a periodical form, either intermittent or remittent. In the continued form, the characteristic lesions of true typhoid fever are found, and in the periodical type the early manifestations are those of malaria, with later development of the typhoid element. It is asserted that the severe form of mountain fever is more liable to prove fatal than typhoid fever in lower altitudes; that treatment is less effective in such elevated regions than nearer sea level, delirium, stupor, and extreme destruction of tissue in the intestines rapidly advancing to a fatal issue. Dr. Hayes, formerly of Denver, Colorado, several years ago reported success in the treatment of this fever with *echinacea*.

SECTION III.

CONSTITUTIONAL DISEASES.

I. RHEUMATISM.

Definition.—A constitutional disease, characterized by pain and tenderness in the locomotor apparatus, including the joints and muscles, with tendency to endocarditis and acid sweats.

Etiology.—Considerable confusion exists regarding the etiology of rheumatism. The latest theory is that it is due to the presence of microörganisms in the blood, and some authors class it as a specific infectious disease. But, while various microbes have been found in the blood of rheumatic persons, there does not seem to be any one that is constantly present. *Defective assimilation* has been ascribed as the cause by Prout and his followers, upon the ground that lactic acid or one of its compounds results from the faulty appropriation of the food, and that this irritates the various tissues of the locomotor apparatus. However, clinical and therapeutical experience has proven this theory to be wrong, as acids instead of alkalies sometimes prove curative, and, in the majority of cases, simple vegetable agents prove more curative than either acids or alkalies. Many regard the disease as a *catarrhal* condition, the causes which produce colds with irritation of the pulmonary or other mucous membranes being directed to the locomotor apparatus instead, and provoking the various unpleasant effects observed here. Such disturbances may be trophic in character, from impressions reflected from the central nervous system; or, they possibly originate morbid secretions, such as lactic acid, through influences exerted upon the sympathetic. One fact is established, and that is, that the disease prevails to the greatest extent in temperate, humid sections, where sudden changes of temperature from warm to cold are common—where exactly the conditions prevail which predispose to catarrhal affections. While rheumatism is more common in England and Canada than in the United States, it grows less common here as advance is made toward the equator, and, in such dry and elevated regions as Arizona, severe cases are almost unknown. It is quite common along the sea coast of central California, but much less so in the more arid regions of the southern interior.

There seem to be certain predisposing causes, such as debility from over-work, bad food, and other unsanitary conditions, tending to bring it on upon slight provocation. Malarial attacks are liable

to be complicated with rheumatism, and rheumatism may follow an attack of malaria and prove very stubborn, the malarial anæmia becoming much aggravated by the rheumatic condition.

The class of persons affected oftenest is that which comprises the robust and middle-aged male population, which is exposed most to vicissitudes of weather. Still, every age and condition may suffer from it, though the disease is rare among very young children. Laborers, sailors, drivers, bakers, iron-workers, and others liable to sudden chilling of the surface when over-heated, or to wetting in the cold rain, are those most commonly subject to acute rheumatism. Heredity is believed to exert a certain influence, it being observed that the members of certain families are especially prone to rheumatic attacks, this probably being due to hereditary susceptibility. The disease presents itself in various forms, and the following varieties will be considered separately:

Acute Articular Rheumatism.

Synonyms.—Acute Rheumatism; Inflammatory Rheumatism; Rheumatic Fever.

Definition.—An acute, non-contagious fever, characterized by severe inflammation and swelling of one or more of the joints, with puffiness and tenderness, and tendency to metastasis.

Etiology.—This has already been sufficiently discussed under the general head. Inflammatory rheumatism usually occurs during the spring and winter months, when dampness and sudden changes prevail.

Pathology.—The changes which occur are not especially characteristic of this disease, more than of any other inflammatory condition. In many cases where there has been remarkable enlargement of the joints and excruciating pain, no *structural change* can be detected after death. In other cases, there are hyperæmia and enlargement of the synovial membranes and ligaments, with turbidity of the synovial fluid, which contains leucocytes and third corpuscles. If the heart be involved, the ordinary conditions of carditis are found, and other inflammatory complications present the usual appearances of inflammation of this part. There is an unusual amount of fibrin in the blood. Suppuration of affected parts is rare, unless there be secondary complications, such as pleurisy, pericarditis, or periostitis. One attack predisposes to subsequent ones.

Symptoms.—There may be anorexia, dyspepsia, *malaise*, and wandering pains, for two or three days prior to the actual onset, though these are often absent, the attack then being abrupt. A chill or, more commonly, *chilly sensations* announce the commencement of

the attack. The temperature now rises quickly, the thermometer indicating an elevation of from 103° to 104° F., within twenty-four hours; the tongue is coated, there are headache and often pain and soreness in the throat. The pulse is full and soft, and running at 100 per minute. The skin is often, though not always, moist and frequently covered with a sour sweat. The urine is scanty, and, on standing, it deposits urates abundantly. Miliaria often appear upon the surface, and sometimes a pronounced roseolous eruption is present. Simultaneously with the onset of the fever, changes in one or more of the joints appear. There may be swelling and puffiness at first, without redness or pain, but pain soon becomes excruciating, and the swollen part is reddened and exceedingly tender to the touch, the weight of the bedclothes being oppressive. The large joints are most apt to be involved, such as the knee, ankle, shoulder, wrist, and elbow, though the fingers and toes may be implicated. Sometimes nearly all the joints of the body may be involved, even the vertebral articulations, the sterno-clavicular joint, the synchondroses of the ribs and symphysis pubis, and the sacro-iliac synchondrosis also. The joints of the arytænoid cartilages have been thus affected.

A marked feature is a tendency to subsidence of the inflammation in one joint, with simultaneous appearance of swelling, pain, and redness in another (metastasis).

Anæmia rapidly develops as the disease continues, and the acid sweats become neutral or alkaline. Endocarditis is liable to develop, an apex *bruit* being now detectable.

The fever declines by gradual lysis in favorable cases, though the disease may continue for weeks when badly managed, permanent stiffness and deformity of the joints remaining as a result of inflammatory deposits about their structures.

In malarial districts, a marked periodicity may become manifest, the pain being paroxysmal, or marked exacerbation may occur every day, or every second day.

There is usually little mental disturbance, the patient being conscious and rational, and thus capable of appreciating his sufferings intensely, unless free use is made of opiates, in which event delirium may be present. Sometimes internal organs other than the heart are involved, the bladder sometimes being severely affected, producing dysuria, with severe tenesmus, or complete ischuria requiring catheterization.

Diagnosis.—The severe joint-symptoms, with tendency to metastasis, will distinguish this disease from others. Pyæmia, where the joints are affected, may resemble it at first, but suppurative synovitis follows in pyæmia, and not in acute articular rheuma-

tism. Arthritis, not rheumatic in character, remains persistently in one joint, while metastasis occurs in rheumatism, more than one joint is apt to be involved, and cardiac symptoms may develop.

Prognosis.—Properly treated, few cases of rheumatism ought to result fatally, and perfect use of the joints ought to follow recovery. The principal danger is in cardiac complication, and this can usually be controlled promptly by Eclectic methods. The disease usually lasts three or four days in one joint, and it may continue three or four weeks, in obstinate cases, though it will usually subside much earlier. Ulcerative endocarditis sometimes remains after an attack of rheumatism, and fatal results follow at a more or less early date. Endarteritis, pleurisy, pneumonia and other pulmonary affections, meningitis, and peritonitis may follow as sequelæ. Chorea also occasionally develops, while subacute or chronic rheumatism may remain after the acute attack has passed off.

Treatment.—We possess a number of effective remedies for inflammatory rheumatism, and only need to adapt them correctly to be speedily successful, in the majority of cases. The *alcoholic vapor bath*, or what is better when it can be obtained, the *cabinet vapor bath*, is excellent and will often succeed alone in effecting a perfect cure in two or three days' time. The application should be thorough enough each time to promote profuse perspiration, and should be repeated every day, and aided, when practicable, by the tonic *faradic* treatment. To assist this, or as an independent measure, two full doses of specific *jaborandi* will be found excellent, from twenty to thirty drops being given two hours apart, the following prescription being administered every hour afterward until recovery, unless it becomes necessary to abandon it in two or three days for other means: ℞ Sp. m. jaborandi ʒiii, water ʒiv. M. Dose, a teaspoonful.

If there be any special indication of blood depravation this should be met in the meantime, in order that special treatment may not be embarrassed. Sometimes there is excessive acidity of the stomach with septic complication, indicated by the creamy, or dirty-white coating on the tongue, and *salts of sodium* may be demanded. Here we may derive benefit from the *salicylate of sodium*, using a three-grain capsule every three or four hours. Usually, however, the *sulphite of sodium* will correct the septic condition better, though it is not so specifically adapted to the rheumatic condition. Occasionally, there may be lack of acids, indicated by the dark red mucous membrane and slick tongue, calling for twenty-drop doses of dilute *muriatic acid*, to be repeated every four hours until the specific condition has been corrected.

When such measures fail to effect a speedy impression, the best

remedy to rely upon is *rhamnus californica*, wine-glassful doses of a strong decoction of the bark, or twenty- or thirty-drop doses of the extract being administered every three or four hours until free catharsis is established, and afterward continued in small doses, just short of catharsis. This remedy will seldom fail to bring about satisfactory results in a few days. A combination which has afforded me good results in past time is: ℞ Sp. m. cimicifuga ʒii, wine of colchicum seed ʒss, spts. nit. dul. ʒi, simple elixir, ad. ʒiv. S. Take a teaspoonful every three hours. *Phenacetin* sometimes brings relief, and is worthy of trial in stubborn cases. A capsule consisting of phenacetin gr. iii, caulophyllin gr. 1-10th, and arseniate of quinia 3x gr. ii, is my favorite form for administration, one being the dose, to be repeated every two hours until profuse perspiration follows.

The general propositions which apply to the treatment of fevers may be applied in the treatment of rheumatism. The irritable stomach will call for *aconite* and *rhus tox.*; periodicity for antiperiodic doses of *quinine*, etc.

Opiates should generally be avoided, as their action is calculated to prolong the disease and increase the liability to serious cardiac complication.

Blisters should be employed sparingly, if at all. Sometimes, when severe cardiac complication seems to threaten vital action, a large fly-blister, applied to the left pectoral region, may produce a desirable derivative effect.

Local applications to the inflamed joints may sometimes be of satisfactory service, and, as the sufferer will usually demand them, they must not be forgotten. Diluted chloroform is probably the best application, an ounce to four of alcohol being used to moisten wrappings of cotton, which should be covered with flannel bandages wrung as dry as possible from hot water. A favorite application with old school physicians, though not very effective, is turpentine, applied freely, the parts being afterward enveloped in raw cotton. Various anodyne liniments have their respective advocates.

The *diet* should be carefully regulated, meat and stimulants being strictly prohibited during the febrile stage. A milk or bread-and-milk diet is sufficient during this time, enough being allowed to satisfy the demands of the appetite; or, if there be anorexia, milk should be administered in small quantities every two or three hours. When milk cannot be taken (and many spleen against it), soups and broths may be allowed instead. Oyster soup, clam broth, oatmeal gruel, etc., are appropriate substitutes. Such drinks as vichy or seltzer may be taken, though plain water is proper, or, where

craved by the patient, lemonade, dilute celery phosphate, barley water, or rice water may be drunk freely. It is well to avoid saccharine food, both during the disease and during convalescence, the diet of convalescence being restricted, at first, to rice, arrowroot, oatmeal, corn meal, unsweetened puddings, soup, wine jelly, blanc-mange, and malted foods. The return to animal diet should be gradual, and only after the fever has subsided for at least a week. Then the yolks of eggs, boiled an hour (one each day), fish, sparingly at first, oysters, and the white meat of broiled or roasted chicken may be taken, along with cooked celery, spinach, asparagus, etc. Baked apples, or pears, without sugar, may be allowed, but sugar and alcohol should be avoided for weeks, as they are liable to provoke a relapse.

Subacute Articular Rheumatism.

This form of rheumatic disease may follow an attack of acute rheumatism, or it may occur in an individual who has formerly suffered an acute attack and afterward been exposed to some exciting cause of the trouble. The pathology is similar to that of the acute form, except that the joint affection leaves no trace of disorganization, though there are similar blood changes. There is not the morbid tendency to metastasis that characterizes acute rheumatism, and the joints do not become reddened and swollen, nor are they painful, unless moved or strained. Only one or two joints may be involved. Anæmia is a manifest symptom, and cardiac complications are liable to occur. The disease may come on gradually, in some cases, without a previous history of acute rheumatism. There is little or no fever. The condition may persist for from six or seven weeks to three or four months.

The *treatment* consists in the steady use of *rhamnus californica*, and the daily application of thorough massage about the affected joint or joints. The rhamnus californica should be prepared by boiling a drachm of the recent bark for twenty minutes over a slow fire (after it has been infused in a pint of cold water), from one to two tablespoonfuls being administered four or five times daily. Where but one joint is affected the patient may be able to apply the massage himself, and then he should knead the affected part vigorously and thoroughly, several times each day. This will be found very effective.

The diet should be unstimulating, milk being preferable. Persons subject to rheumatism should wear flannel underclothing throughout the year.

Chronic Articular Rheumatism.

Synonym.—Chronic Rheumatism.

Definition.—A chronic, articular disease of advanced life, characterized by thickening of the capsules and ligaments of the joints, without marked deformity, the disease being aggravated by dampness and atmospheric changes.

Etiology.—This is a disease which comes on after middle life, either insidiously or as a sequela of former attacks of acute or subacute rheumatism. Damp localities predispose to it, such as dark and damp dwellings, sleeping in ground-floor apartments, or over damp cellars, etc. It is aggravated during the winter and spring, and the joints are rendered stiff and lame from prolonged rest, motion and exercise tending to relieve them for a time. It is most common among those of laborious occupation. Such persons are usually good "weather prophets," as they are susceptible to atmospheric changes, and are usually influenced in advance of the advent of a marked change of weather. Sometimes only one large joint may be affected, though several are usually involved at the same time.

Pathology.—In some cases there is no structural change, the synovial structures being injected, but not much altered otherwise, there not being much effusion. Usually, in long-standing cases, the fibrous tissue around the joints, the fibrous envelope of the nerves, the fasciæ, the peritoneum, and the aponeurotic sheaths of the muscles are all involved in chronic inflammation. There is thickening as well as increased vascularity of the synovial membranes, the fringe-like processes are enlarged, and the synovial fluid is turbid. Sometimes there are erosions of the articular surfaces. Deformities may arise from the formation of constricting bands of fibrous material about the diseased joints, and the deformity may be more prominent from atrophy of the surrounding muscles, through local disease or from reflected trophic influences. Cardiaccomplications are seldom present.

Symptoms.—The symptoms come on gradually, slight soreness and stiffness of the affected joints being first noticed during damp and cloudy weather, or on the day following some severely laborious occupation. This is more noticeable upon rising in the morning, and it gradually disappears as the affected part becomes accustomed to action. The affection becomes more troublesome during sudden changes, and in the cold and damp months of winter and spring. The joints gradually become more impeded in their range of motion, finally being painful when at rest. There is usually slight tenderness upon pressure, but the joints are

only slightly swollen and not reddened, unless there be extreme aggravation. Suffering is usually increased at night and ameliorated by exercise in the morning. Stiffness, soreness, and impairment of the joints slowly increase with advancing age, though there is never marked deformity nor serious loss of motion. The large joints are usually involved, though the finger-joints may be affected in those who use the hands severely, such as washerwomen.

Diagnosis.—This affection should not be confounded with arthritis deformans, for here there is marked deformity of the joints, almost complete loss of motion, and gradual progress from one joint to another, with never any improvement, while weather changes exert no influence upon it. There is also pronounced deformity in gout, and the small joints are the parts affected.

Prognosis.—While not inimical to longevity, the prognosis, as to a cure, is not favorable, unless the patient be removed to a new and healthful climate. However, when proper treatment is begun early and persevered in, the severity of the disease may be much modified. After structural changes have gone on in the joints, palliation of the most distressing symptoms is the best that we can hope for without radical change of climate.

Treatment.—Early in the disease, *massage* and *electricity* will be of good service. Both faradism and galvanism are useful, strong currents of each being passed through the affected part or parts, alternated at every sitting (every two or three days).

Rhamnus californica is now of some use, and it should be persisted in for months, alone, or combined with *grindelia squarrosa* if the neighborhood be malarious. The patient should *dress* the year round in warm flannels, to prevent chilling of the joints from draughts and dampness, and avoid a *calling*, so far as possible, requiring much outlay of muscular effort. The *diet* should be nutritious, but red meat should be generally avoided, the patient being instructed to depend upon fish, eggs and fowl, avoiding sweets and alcoholic drinks, the basis of his diet consisting of farinaceous food, with a few fresh vegetables.

Aggravations should be met appropriately. Periodical aggravations will call for proper antimalarial treatment. Active inflammatory aggravation of a particular joint may be benefited by blistering the part. Chloroform liniment may relieve the pain at night. In other cases, warm or cold applications afford more relief.

Patients of competent means should be advised to spend their winters in a warm climate, southern Europe, southern California, or Arizona offering prospects of greater comfort than ordinary climates. There are certain thermal springs that afford much benefit to these

patients, such, for instance, as the Hot Springs of Arizona or Virginia, Byron Springs, near San Francisco, or those of Banff, in the Rocky Mountains, etc.

Muscular Rheumatism.

Synonym.—Myalgia.

Definition.—A painful disease of the muscles and their fasciæ, as well as of the periosteum, arising from constitutional influences.

Etiology.—Sudden chilling after exertion, overstrain of the muscles, protracted exposure to dampness, and malaria are among the exciting causes. It is most commonly met with in those who apply themselves to severe bodily exertion, and is apt to follow draughts of air, wetting from chilling rains, etc. Rheumatic or gouty persons are most subject to attacks, and they may be seized during changeable weather, without undue exposure.

Pathology.—Investigations into the pathology of this disease have thrown little light upon the subject. The muscles undergo few if any anatomical changes, and such changes are not constant. Occasionally there may be evidence of inflammation of the sheaths of the muscles, or scanty serous exudation into their substance, and, at other times, signs of degeneration of the muscular fibers. Sometimes thickening or degeneration of the neurilemma of the nerves supplying the part may be observed.

Symptoms.—Many attacks of muscular rheumatism occur suddenly. "Crick-in-the-back" is an illustration of this, the person, in apparently the best of health, being suddenly seized with an excruciating pain in the lumbar region, so severe as to give rise to intense suffering upon the least attempt at motion. Severe attacks of pleurodynia also occur, in which the body is drawn toward the affected side, and breathing is accompanied by intense, lancinating pains. In malarial regions, muscular rheumatism is very apt to be periodical, the attacks occurring in regular exacerbations, every day or every second day, with remissions or complete intermissions between. Such attacks may pass off in a few days, or may turn into a chronic form, the pain becoming permanently located in some muscle or group of muscles and causing almost constant discomfort. Sometimes the muscular structures of the internal organs, such as the stomach, intestines, bladder, œsophagus, etc., may be involved, the functions of these parts then being impaired, while a painful state of the part exists simultaneously. The disease is almost purely local, little febrile or other systemic disturbance being present.

Localization of the pain in various regions has given origin to a number of special names for this affection, such as lumbago (lumbar

rheumatism), torticollis (stiff neck), and pleurodynia or rheumatism of the intercostal or other muscles of the chest, those of one side usually being involved. Sometimes the abdominal muscles are distinctly involved, giving rise to severe cramping pains in this region.

Diagnosis.—The characteristic symptoms of muscular rheumatism will usually distinguish it, *metastasis* being a symptom not common to any other painful disease of the muscles. The periosteal pains of syphilis will be distinguished by the fact that changes of weather do not affect them appreciably, and the accompanying symptoms of syphilis will afford additional light on the subject. *Lumbago* might be mistaken for renal colic, but it is to be remembered that lumbar pain in lumbago renders motion painful and difficult, while in renal colic the patient moves about in all positions during the paroxysm, and the pain darts along the ureter of the affected side, the corresponding testicle often being retracted, while the urine is scanty and probably bloody. In spinal pain, pressure upon the spinous processes causes increased suffering while lateral pressure is not painful, the opposite being the case in lumbago. *Pleurodynia* is often diagnosed as pleurisy. It should be remembered that pleurisy is attended by fever and cough with friction sounds on ausculation, while in intercostal rheumatism the principal symptoms are pain and dyspnœa, while motion of the affected muscles aggravates. *Abdominal* rheumatism may be mistaken for peritonitis, but the absence of fever and the severe constitutional symptoms attending this disease will clear up any obscurity. *Trichinosis* is attended by pains which resemble those of muscular rheumatism, but here there is œdema of the feet, and the history of the case, usually occurring in several individuals simultaneously, with microscopical examination, will settle any question of this character.

Prognosis.—There is no danger of a fatal termination of this disease, though it is quite liable to return upon slight provocation. An acute attack may be relieved in a few hours, though when neglected it may become chronic and prove very troublesome.

Treatment.—To relieve an attack of muscular rheumatism, the alcoholic or steam *vapor bath* answers an admirable purpose. Sometimes an extremely severe case may be advantageously relieved, in the start, with a 1-4th grain dose of morphine, either hypodermically or per mouth, though the use of opiates is, as a rule, to be avoided, as they seem to finally fix the disease in the system and render it more stubborn. The vapor bath may be assisted by two or three wine-glassful doses of a hot decoction of *cimicifuga* root, taken every twenty minutes or half-hour.

Periodicity should be expected in malarious regions, and a return

of the attack be anticipated within one or two days, appropriate doses of quinine or arseniate of quinia being administered.

As a specific remedy, *cimicifuga*, in tablespoonful doses of a cold decoction, may be continued for several days, every three or four hours, or, where desirable to employ smaller doses, the specific medicine or a saturated tincture of the root may be used, by adding half a drachm to four ounces of water. A combination of aconite and cimicifuga (Scudder) answers well, the prescription being as follows: ℞ Lloyd's aconite gtt. v–vii, green plant tincture cimicifuga gtt. xx, water ʒiv. M., and order a teaspoonful every hour.

Where cimicifuga fails, where the tongue is coated yellow, or where there is habitual constipation, *rhamnus californica* may be used, a decoction (ʒi to the pint of water), in double-tablespoonful doses, being preferred to any alcoholic preparation. The dose should be reduced if too free action on the bowels follows.

When the pain becomes localized and remains stubbornly, the following prescription, continued for a fortnight, will often result in a cure: ℞ Phenacetin gr. iii, caulophyllin gr. 1-10th, arseniate of quinia 3x gr. ii. M., ft. capsule no. 1. Duplicate no. 60. Sig., Take one every four hours. The prolonged use of this capsule may result in profuse and and prolonged perspiration, the subsequent use of two-grain doses of picrotoxin 3x, four times daily, being required to control it. *Massage* is of much assistance in chronic cases, the muscles gaining tone and energy under its influence, and being thus enabled to better resist disease agencies.

In chronic cases, we will rely upon this agent in connection with the prolonged use of *rhamnus californica* or *manaca*, occasional alternation of these remedies being advisable. Manaca may be administered in from two- to five-drop doses (fl. ext.) four or five times daily.

In malarious districts, much advantage will attend the continued use of fluid extract grindelia squarrosa (P. D. & Co.), in ten- or fifteen-drop doses, three or four times a day, in connection with the antirheumatic.

The diet should be similar to that recommended under the treatment of articular rheumatism.

II. PSEUDO-RHEUMATIC AFFECTIONS.

Arthritis Deformans.

Synonyms.—Rheumatoid Arthritis; Rheumatic Gout.

Definition.—A progressive, destructive disease of the joints, characterized by inflammation and degeneration of the cartilages and synovial membranes, with the development of bony growths

upon the articular surfaces, and thickening of the ligaments and other soft parts, rendering the joints immobile and deformed.

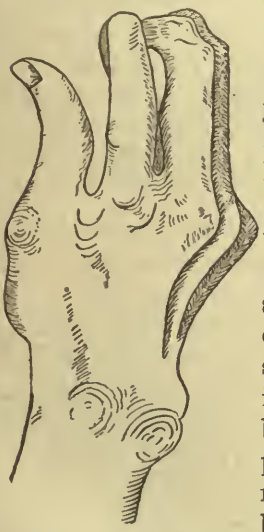
Etiology.—Though formerly believed to be closely related to both rheumatism and gout, there is a rapidly growing belief, among pathologists, that no connection exists with either, in the causes or nature of this affection. The pathology differs essentially from both that of rheumatism and gout, there being no blood changes as in rheumatism, and no urate of soda deposits as in gout. The symptoms, in the start, may so closely resemble those of chronic articular rheumatism as to render the distinction difficult, but this is probably a matter of diagnostic obscurity rather than of similarity of nature. It is asserted that several facts tend to confirm a nervotrophic theory. Of these may be mentioned the similarity of the affection to joint diseases due to affections of the cord, as in locomotor ataxia; the apparent origin of the disease from shocks, mental worry, etc.; the tendency to symmetrical distribution of the lesions, and changes in the muscles, skin, and nails, evidently due, in great measure, to trophic influences. It is asserted that damp dwellings and insufficient and improper food predispose to it, though it seems to be an assumption rather than an established fact. Heredity may exert some influence, though the disease is so rare that a family record over a protracted period must be necessary to arrive at definite conclusions. It is more common in females than in males, and the small joints are most liable to be involved in this sex, males being more subject to involvement of the large articulations. The elderly, the middle-aged, and the young may be affected, though the period between twenty and thirty is the age most susceptible.

Pathology.—The articular cartilages and synovial membranes suffer the earliest and most marked changes, though the ligaments and muscles undergo prominent alterations later on. The articular cartilages become softened and villous in the center, where the greatest pressure is exerted, and gradually wear away, until the extremities of the bones are exposed, these then becoming eburnated and constituting the articular surfaces. The circumferences of the cartilages remain and undergo irregular nodulated proliferation, ossification setting in later, the bony nodosities (osteophytes) serving to lock the motion of the joints. Meantime, the fringes of the synovial membranes become increased in number and hypertrophied, from augmented vascularity, and later undergo fibrous degeneration, the whole membrane thus becoming thickened and hardened. New bone may also spring up from the periosteum, and the joint gradually becomes locked with bony growths, and firmly bound with thickened bands, the ligaments, as well as the synovial membranes, becoming

hardened and thickened, until the joint is finally almost immovable. The muscles atrophy at length, and both hypertrophy and atrophy occur about the expanded extremities of the bones, varying deformities thus arising. The nerves about the joint may participate, a chronic neuritis becoming established.

Symptoms.—This disease may be divided into two general forms, viz., acute and chronic. The acute form may be divided into two varieties, the nodosities of Heberden, and the general progressive form.

The *nodosities of Heberden* are usually confined to the fingers, the disease becoming arrested after involving these parts. The tubercles at the sides of the dorsal surfaces of the second phalanges become slowly enlarged, slightly reddened, and the affected joints are easily hurt by accidental knocks, though not usually painful. The cartilages may become soft, and the extremities of the bones bared later. The joints gradually become more and more stiffened and disfigured, until all use of the affected part is lost, the patient meantime enjoying good health otherwise. This form is most common among women.



Arthritis Deformans.

The *general progressive form* may come on suddenly, with acute symptoms, or it may develop by a chronic course. Acute attacks simulate subacute articular rheumatism, though it will be observed that there is absence of blood changes, acid sweats, and cardiac complication. There is swelling, pain, and soreness of the joints, the synovial capsule and bursæ being especially involved, redness usually being noticed, and the local symptoms are accompanied by moderate febrile disturbance. Periods of recuperation and exacerbation may attend this form, the acute symptoms subsiding after a few weeks or months, and an approach toward recovery apparently following, to be succeeded by relapses or exacerbations until the disease has progressed so far as to hopelessly cripple the subject. Anæmia, followed by slight hectic, may ensue upon the subsidence of the fever, the disease thus insidiously advancing, the patient never entirely recovering complete use of the joints

Children, or women between twenty and thirty years of age, who have become debilitated by child-bearing or from excessive lactation may be affected, the puerperal period being an apt time for the acute onset. It may also occur about the menopause.

The *chronic* form of progressive arthritis deformans comes on insidiously. Slight pain, tenderness, and swelling may involve a single joint at first, apparent recovery shortly ensuing, perhaps, but recurrences continue to follow, one after another, until permanent deformity and impairment of function become settled. Other joints have become involved before this time, sometimes all the large ones being implicated, though often not more than one or two may be affected. These gradually become rigid and motionless, and muscles atrophy from disuse and degenerative changes, contraction of the flexors gradually drawing the thighs upon the abdomen and the legs on the thighs, while the arms are drawn to the sides with the forearms flexed upon them and the articulations locked, the patient remaining in a recumbent posture, unable to move about or use his extremities, except, possibly, the hands, which may have escaped.

Diagnosis.—There is more deformity and less severe pain when the joints are at rest than in acute or chronic rheumatism, and immobility progresses more rapidly than in either of those affections. In gout, the smaller joints are almost exclusively affected, while in this disease all joints are equally liable, and the attacks are not erratic, as in gout.

Prognosis.—There is little prospect of recovery, after the disease has progressed far enough to render the joints immobile, though if treatment be begun early it may be considerably modified.

Treatment.—We know so little about remedies which influence trophic impulses that we cannot prescribe with any certainty on these lines. However, we are acquainted with a few remedies which seem to exert an influence over the nutrition of the joints, and it is possible that some if not all of them act through the trophic centers. It is certain to my mind that minute doses of *silica* 3x, continued through a long period (a year or more), exerts a decided influence upon arthritic conditions of the finger-joints (Heberden's nodosities). With this as a pointer, we may make a systemtized study of such other agents as have been known to favorably influence joint affections of various character, and, as the chronicity of these cases will afford a good opportunity to test them well, they may be used in rotation, giving each one an extended trial, unless, perchance, the successful one should be found early in the day. We will find, upon referring to Dynamical Therapeutics, that *calcium floride*, *stillingia*, *berberis aquifolium*, *corydalis*, and several other remedies are serviceable in arresting the development of nodes, while *cistus canadensis*, *ledum palustre*, and *pulsatilla* possess the reputation of influencing various structures about the joints. A careful study of these remedies in this connection will afford some satisfaction to the investi-

gating physician, and may prove of lasting benefit to the patient. *Massage* is an excellent measure, and it should be put in practice early and persisted in for years, especial attention being paid to the spinal column, joints, and muscles. Cold compresses are serviceable to relieve pain in the joints, and should be applied at night especially (provided they are comforting to the patient), that rest may contribute toward recovery. Motion of the joints should be avoided, except passive motion during massage to prevent contraction of the muscles. Electricity affords little if any benefit. Depleting agents, such as iodide of potassium and the salicylates, should be avoided.

The *diet* should be generous, nourishing, and stimulating—the very opposite to that of rheumatism. Where the digestion is good, beefsteak, roast beef, mutton, and fowl, fish, eggs, and milk may be taken liberally. Also fats, such as butter, cream, suet pudding, olive oil, and other oleaginous articles. Malt liquors are not objectionable here, and porter, ale, or stout may be used, to stimulate digestion, and assist in nourishing the patient.

Where the patient is financially qualified, the hot springs of Virginia, Arkansas, or Banff may be recommended—before the disease has advanced beyond reasonable prospects of benefit.

Gonorrhœal Rheumatism.

Definition.—A septic synovitis or arthritis, due to infection from gonorrhœal virus.

Etiology.—It is now generally believed that ptomaïnes generated within and absorbed from the urethra give rise to the mild or non-suppurating form, while the more severe or suppurating cases arise from the infection of the system with pus-organisms. It occurs more frequently in men than in women, possibly because the genital passage affords greater opportunity for the burrowing of the gonorrhœal virus. Relapses are common and progress slow, under the most favorable conditions.

Pathology.—There is synovitis, with dryness of the synovial membrane, a crackling sound attending motion of the joint. In severe cases, there is destruction of the cartilages, and permanent thickening of the synovial membrane.

Symptoms.—The symptoms vary considerably, the disease sometimes running an acute and rapid course, and at others assuming a chronic condition, which may last for years. In some cases, the symptoms will be limited to arthritic pains, which linger about the joints for a long time, there being total absence of redness, swelling, or tenderness. Sometimes the joints are intensely painful, red-

dened, and swollen, the condition resembling inflammatory rheumatism, but being of more persistent and less active character. In another case, a single joint may be involved, with extreme swelling and œdema and a probability of suppuration, though resolution may follow. Chronic hydrarthrosis may be one of the conditions, while in other cases the bursæ of the patellæ, olecranon, and tendo Achillis may be the parts principally affected.

One peculiarity about the disease is its tendency to involve articulations seldom affected by articular rheumatism, such, for instance, as the sterno-clavicular, sacro-iliac, intervertebral, and temporo-maxillary. Pain is a prominent feature.

Diagnosis.—The history of the case will be sufficient aid in diagnosing this disease from other affections of the joints.

Prognosis.—Not favorable to a speedy termination, though it may not shorten life.

Treatment.—*Berberis aquifolium*, *cistus canadensis*, and *echinacea* should be tried persistently. *Cabinet vapor baths* afford some relief. It is asserted that free incision of badly affected joints with subsequent irrigation, affords the best results. *Fixation* of the joints to prevent motion alleviates much pain.

III. GOUT.

Synonyms.—Podagra.

Definition.—A disorder arising from disturbances of the assimilative functions, characterized by attacks of acute inflammation of the small joints, with the gradual deposition of urate of soda in the articular cartilages and other parts of the joints, with erratic constitutional disturbances.

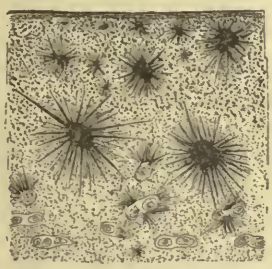
Etiology.—Sixty per cent of all cases of gout are hereditary, showing a constitutional predisposition. Inability to properly assimilate nitrogenous material results in an excess of urates in the system, these becoming deposited in the cartilages and other tissues, and exciting inflammatory action.

Gout is a disease of middle life, few suffering from it while young, unless there be an exceptionally strong hereditary tendency. The gouty person is diseased from inability to oxidize nitrogenous food; excess of this, faulty digestion, and sedentery habits—which tend to lack of oxygen inthe system— being provoking factors. Workers in lead are especially prone to gout.

The idea that all gouty persons are gourmands is a mistaken one An individual may be an apparently moderate consumer of nitrogenous food and yet be gouty, because he peculiarly lacks the consti-

tutional ability to safely dispose of nitrogenous food, either through sedentary habit, hereditary weakness, or character of vocation. Not all subjects of gout are wealthy and high-lived. The disease occurs among the poorer classes who consume much malt liquor, in connection with poor food and bad hygienic surroundings.

Pathology.—The blood of gouty persons shows an excess of uric acid. If five or six drops of acetic acid be added to ʒiii of blood-serum from a gouty person in a watch-glass, and a thread be immersed here a few hours, it will be found to be incrusted with crystals of uric acid. The same results occur, however, when serum from the blood of a leukæmic or chlorotic individual is employed. In gout, the uric acid combines with sodium, and becomes deposited as urate of soda in the tissues of the articulations, especially of the lower extremities.



GOUTY CARTILAGE.

PAPILLA OF GOUTY KIDNEY.

These deposits may become extensive in chronic gout and form concretions of chalky material (tophi or chalk-stones), which, in extreme cases, may even cause ulcerations through the skin, and appear externally. These concretions, in greater or less quantity, accumulate in the articular cartilages and cause necrotic areas, the part furthest from the circulation being probably most affected. The cartilage in the first joint of the great toe is liable to be involved in the beginning, but the knees, ankles, and small joints of the hands become affected in succession. The cartilages are first involved, then the fibro-cartilages and ligaments may be infiltrated; the synovial fluid may also contain crystals of urate of soda. Immobility of the joints results in long-standing cases, as exostoses, occurring upon the margins of the articular surfaces, serve to lock those already stiffened by concretions deposited in the fibro-cartilages and ligaments. Chronic gout is often signalized by the appearance of yellow nodules in the cartilage of the ear, at the margin of the helix, composed of gouty concretions. These may also accumulate in the cartilage of the eyelid, nose, and larynx. The deposit is interstitial,

though it may appear to be upon the surface of the cartilage, subsequent coagulation necrosis affecting its release.

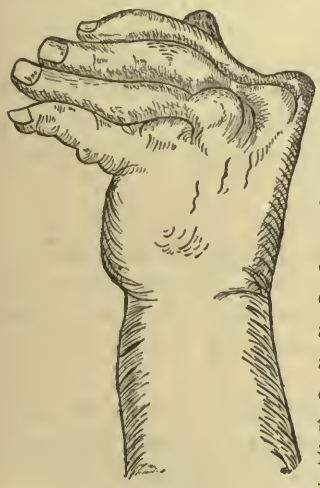
The kidneys and arteries suffer most severely, after the joints. Both cortical and medullary portions of the kidney may be infiltrated with crystals of urate of soda, but the papillæ are most prominently affected, striæ of whitish deposit appearing here, both in the intertubular tissue and within the tubules. These occur in interstitial nephritis, and their presence cannot be considered pathognomonic of gout unless there be the articular disturbance to confirm it. Arterio-sclerosis commonly occurs in chronic gout, it being asserted that concretions of urate of soda are found on the cardiac valves, the left ventricle being hypertrophied.

Symptoms.—Gout may be divided into regular and irregular forms. In regular gout the manifestations occur about the joints, principally, while in irregular gout there are no arthritic manifestations, internal organs being the points of uratic deposit. In a few acute cases there is *retrocedent* or *suppressed* gout, the disappearance of the arthritis being attended by serious disturbance of internal organs, such as severe gastro-intestinal symptoms (vomiting, purging, abdominal pain, and prostration), cardiac manifestations, such as dyspnœa, irregular action of the heart, or angina, and sometimes cerebral complications, such as delirium and coma, or apoplexy.

In *acute gout* there may be premonitory symptoms, such as fugitive pains in the small joints of the hands and feet, insomnia, indigestion, and irritability of temper, for several days prior to the onset. If the urine be examined now it will be found to contain urates and traces of albumin or sugar. Asthmatic attacks may also occur during this time. At length, between the hours of one and four o'clock A. M., the subject is suddenly seized with an excruciating pain in the metatarso-phalangeal articulation of the great toe of one foot (usually the right), and this persists for hours, the pain being throbbing, tensive, or burning. The part swells rapidly, and soon appears as though suppuration were impending. It is hot, tense, and shiny, and extremely sensitive, the pain becoming agonizing, as though the part were squeezed in a vise. The pain subsides in the morning, though the part remains swollen throughout the day, and about the same hour on the following night there is a recurrence, this state of affairs continuing for six or eight days, the severity of the symptoms gradually wearing away. During the paroxysms there is considerable constitutional disturbance, the temperature rising to 102° or 103° F. Desquamation of the skin follows the subsidence of the swelling. Within from three months to a year another attack occurs, and now the disease is liable to manifest a tendency to reach

further out and involve new territory. If one joint only was affected at first, the corresponding joint on the opposite foot may now be affected also, and another time one or more additional joints may be involved. The recurrences incline to become more and more frequent after each repetition, and, finally, a constant inflammatory condition, constituting chronic gout, becomes established. During all these attacks suppuration of a joint never occurs.

All gout is, strictly speaking, *chronic gout*, but the term is usually applied to those cases where the paroxysms coalesce and the joints have become permanently involved. Concretions of chalky material have now formed around the articulations, and there is such crippling as to interfere with locomotion and prehension. The joints become noticeably distorted—enlarged and nodulated—and immovable, the skin covering them being congested, and the superficial veins dilated. Tophi may perforate the surface here later, and be discharged as a yellowish-white substance, or remain, causing chronic ulceration.



Gouty Hand.

The general health now appreciably deteriorates, the "gouty diathesis" being established. The skin is pale and sallow, and the patient presents a general appearance of invalidism. There are muscular cramps, dyspeptic symptoms, cardiac disturbances with occasional præcordial pains, irrritability and restlessness, nocturnal wakefulness, tic douloureux, urinary difficulties with albuminous deposits, gouty abscesses, etc. Daily heat and redness of the nose is a common symptom.

Irregular gout presents itself in numerous forms. Retrocedent gout, which occurs in acute attacks, is included under this term. Numerous individuals who belong to gouty families may never manifest articular disease, and yet be subject to gouty affections—uratic deposits in structures other than those of the joints—and these may be either acute or chronic.

Nervous affections are common results of irregular gout. Headache, vertigo, delirium and acute mania may result from retrocedent gout. More chronic conditions arise from gradually acquired uratic deposits in internal organs. Epileptic seizures, neuritis, with neuralgia, formication and numbness, startings of the limbs, cramps, meningitis, or apoplexy may be due to the influence of gouty deposits

in various portions of the nervous system, though the apoplexy will usually be due to involvement of the cerebral arteries. Gouty implication of the sheath of the sciatic nerve may give rise to an obstinate form of sciatica, which may extend upward and involve the spinal cord.

Vascular disorders may arise from gouty deposits in various parts of the circulatory organs. There is no special cardiac inflammation arising from gout, as in rheumatism, but cardiac disturbances from gouty encroachment are not uncommon. Patches of deposit may occur upon the pericardium. Valvular enlargement and obstruction, with subsequent hypertrophy and fatty degeneration of the heart-muscle, may arise from uratic deposits in their structure. Atheromatous conditions of the arteries, or arterio-capillary fibrosis with thickening of the muscular coat of the small arteries may be set up. Various unpleasant symptoms may thus arise, such as erratic cardiac pains, and sometimes angina pectoris, palpitation at times, or slow, feeble, and irregular or intermittent pulse, sensations of constriction about the cardiac region, with dyspnœa, anxiety, and sense of impending dissolution.

Digestive disorders are common symptoms of irregular gout. There may be erratic pains in the epigastric region with difficult digestion, or occasional attacks of gastritis, with cramps and vomiting, accompanied, perhaps, with intestinal colic and diarrhœa. Hepatic colic may arise, or fatty degeneration of the liver. Dysphagia is not an unfrequent symptom.

Urinary difficulties are among the complications of irregular gout. There may be renal colic with calculous formations, both renal and vesical, with chronic vesical and urethral irritation, and, sometimes, oxaluria.

Pulmonary affections may be gouty in character, and due to uratic deposits in these organs. Asthma, dry bronchitis, and emphysema, are the common ailments, pneumonia never arising as a sequela of gout.

Cutaneous affections, arising in persons of hereditery tendency to gout, may be very stubborn and intractable, and be ascribed to a gouty diathesis. Eczema, erythema, acne, urticuria, psoriasis, prurigo, both local and general, as well as destructive inflammation of the iris, with loss of sight, may be ascribed to gouty influences.

Diagnosis.—The only disease liable to be mistaken for gout is rheumatism. It will be remembered that gout is comparatively rare in this country, while rheumatism is quite common. The onset of gout is also peculiar, only one joint being involved in the beginning (two at most), and that usually the metatarso-phalangeal articula-

tion of the great toe. The time of attack—between midnight and morning—is also peculiar. Subjects of gout are either hereditarily predisposed, or they have been addicted to high living or malt liquors and are accustomed to sedentary habits, while subjects of rheumatism have been accustomed to hardships and exposure. Acute articular rheumatism affects young persons and is usually attended by high fever, while gout hardly ever comes on before thirty-five and is attended by mild febrile symptoms. Tophi never form in rheumatism and are common in gout, and the microscope will detect crystals of uric acid in the blood of gouty persons but not in those with rheumatism. The joint affection of pyæmia will hardly be mistaken for that of gout, as the previous history will not permit of any confusion of this kind.

Prognosis.—Gout is not a rapidly fatal disease, and its subjects may live to old age, though serious visceral complications are liable to eventually terminate fatally. Recovery is rare, unless the subject abandons a sedentary life and high living. Albuminous urine, with absence of uric acid in the secretions, is ominous, especially if the subject be cachectic and the joints are greatly crippled.

Treatment.—A gouty subject should forever abandon an in-door and sedentary life, and forswear alcoholic liquors and saccharine substances. An out-door life, with moderate exercise, should be adopted, and he should eat to live, not live to eat. A high and dry location is the best place of residence, and a climate permitting of plenty of out-door life the year round is preferable, as this conduces to the inhalation of a generous amount of oxygen. High, well ventilated rooms, without draughts, should be occupied at night, and early retiring should be an established custom. Young persons hereditarily disposed to gout should be encouraged to take plenty of exercise, though actual subjects of the disease should avoid active exertion.

The *diet* is an important consideration. Sugar should be strictly avoided, and fruits containing much saccharine material should also be dispensed with; for, though not a nitrogenous material, the presence of sugar during the digestion of nitrogenous food tends to the formation of uric acid. Sweet wines and malt liquors, for similar reasons, come under this objection. Dilute old claret and whisky, or dry sherry are the least objectionable, though total abstinence is best, unless there is some urgent need for alcohol—something not liable to often occur. Much starchy food is also objectionable, as the starch is converted into sugar during digestion.

The diet of a gouty person should consist principally of fresh green vegetables, with a few fruits—which do not contain much

sugar—used sparingly. As this entails a certain objectionable bulkiness to the amount of food required, however, a small portion of lean beef or mutton may be allowed each day, though this should never be cooked but once—never warmed over. Pickled meats should never be used, as they are especially objectionable, and so also are all forms of pickled fish. Certain kinds of fresh fish are allowable, such as those which are tender when boiled or broiled (the form to be taken in), but those of firm flesh and containing considerable fat, like the salmon, mackerel, halibut, and codfish should be avoided. The best fish for the gouty are the bluefish, whitefish, bass, and shad, which may be taken occasionally, as a change of food. Oysters and clams are allowable, but shrimps, lobsters, and crabs should be prohibited, their use on salads being especially pernicious. Eggs are generally prohibited. Milk disagrees with many, and is believed to be injurious to old persons afflicted with gout. Veal, pork, bacon, ham, and game are not allowable. A few vegetables are prohibited on account of the fact that they contain oxalic acid, which produces oxaluria. These are sorrel, radishes, asparagus, and rhubarb. Some debar tomatoes and spinach, on the same grounds. Beets, being sugar-producing vegetables, are forbidden, and also those which possess the objection that they occasion flatulence, such as cabbage, green corn, matured peas, beans, onions, and corn. Oil should be omitted from salads, as all fats embarrass the digestion of nitrogenous food. All food cooked in fat must be avoided, though butter may be allowed sparingly. Farinaceous food, stale bread, rice, sago, tapioca, oatmeal, and cracked wheat may be allowed, but pastry, hot rolls, warm bread, hominy, and griddle cakes should be avoided. All preserves and confectionery are to be forbidden; also fruits which contain a large amount of sugar, as grapes, figs, banannas, and prunes. Strawberries are also objectionable, because they contain much acid. Fruits, served for the gouty, should be taken without the addition of sugar. Apples and pears, when well-ripened (fresh, baked, or stewed), may be allowed. Oranges and lemons may be partaken of sparingly. Nuts, pickles, vinegar, spices, strong condiments, salted foods, truffles, and mushrooms are to be prohibited.

Fluids are essential, and *water* may be taken freely, as plenty of diluent tends to flush the tissues and wash away gouty material. All fluids, however, should be taken between meals. Weak tea and coffee may be taken without sugar, though they are better omitted entirely, not because they aggavate gouty conditions, but because there are other objections. Alcoholic drinks have already been mentioned. *Saline* and *mineral* waters are excellent for gouty subjects,

and two or three courses at certain mineral springs every year are to be commended. Some of the leading waters of this country are at the springs of Saratoga, New York; St. Clair, in Michigan; St. Catherine, in Ontario; the hot springs of Arkansas, Virginia, and California (Paso Robles), and various others. The Carlsbad Springs in Bohemia are a favorite resort for Europeans and many Americans.

The *medical treatment* of gout consists in the use of *colchicum* and *guaiacum* during acute attacks, and *lithium* as an alternate, or remedy for the intermediate periods. Five grains of the citrate of lithium may be taken at a dose, three or four times daily, dissolved in a glassful of potash water. During an acute attack, the affected foot should be elevated and from twenty to thirty drops of wine of colchicum administered every three hours, until its purgative action becomes manifest, when the dose should be diminished to one-fourth the size, or less. This may be alternated or combined with the citrate of lithium, as already directed. A flannel or cotton batting wrapping should be kept around the affected joint, and this may be moistened with chloroform liniment, to be repeated, if relief attends its action. Sometimes a hypodermic injection of morphine, near the joint, is useful to relieve the severe suffering. Chronic gout will require the judicious selection of proper tonics, the influence of silica upon the nutrition of cartilage being remembered.

Dr. Lional S. Beale believes that other alkalis than lithia are sometimes more efficacious. He believes in their rotation, as the single agent seems to finally lose much of its effect when continued for a protracted period. He thus employs various carbonates, such as carbonate of lithia, potash, soda, and ammonia. Vapor baths will assist in the treatment of chronic cases, by promoting activity of the skin.

During acute gout considerable benefit may be derived from jaborandi and other properly selected sedatives, administered often, in small doses, though as much benefit must not be expected as in rheumatism.

IV. LITHÆMIA.

Definition.—A condition in which there is an excess of uric acid in the blood, characterized by disturbances in the retrograde changes of the body, with excess of lithic acid in the urine, the renal secretion being increased.

Etiology.—This disease is a functional one (there being no uratic deposits in the system), the tendency to such a condition depending largely upon inheritance. It afflicts those who have never indulged in excessive eating or drinking, a primary weakness of the

digestive and assimilative powers seeming to be at the foundation of the trouble. In other cases, excesses in eating or drinking, or protracted sedentary habits may be reasons for its acquirement. Indulgences in the consumption of meat, stimulants, or narcotics, sexual excesses, or prolonged mental or nervous strain may either develop such a state, or aggravate and render prominent latent lithæmic tendencies already inherited. Lack of power to digest nitrogenous material results in the formation of uric acid in the blood. The condition is sometimes associated with rectal disease, especially papillæ and pockets, and it seems as though this condition might be a frequent causal factor.

Anything that interferes with a free supply of arterial blood to the superficial capillaries will naturally tend to bring about lithæmia. Obstruction may be one cause, and perverted or inadequate peristaltic action of the alimentary canal another. Splenic or hepatic engorgement tends to interfere with the free flow of blood through the cæliac axis, and therefore interferes with a vigorous abdominal circulation. Malarial cachexia commonly predisposes to this condition and lithæmia may arise as a result. The second condition, torpid peristaltic action, may arise from chronic constipation, the frequent cause of this condition being rectal disease, with sphincteral spasm. That oxygen in the intestinal capillaries aids in the elimination of uric acid in the blood has been proven experimentally, by the rectal use of oxygen gas in lithæmia.

Pathology.—Arterial changes are the most common and serious pathological changes which occur. While there is no evidence of uratic deposits in the tissues, arterio-sclerosis from fibroid degeneration frequently occurs. Many disturbances similar to those arising from irregular gout also arise, such as gastro-intestinal irritation, asthma, cardiac disturbance, neuralgia, and headache.

Symptoms.—Acute and chronic indigestion are the leading features of this disease. Acid eructations, flatulency, and constipation are usually present, though the bowels may move regularly or there may be diarrhœa alternated with constipation. Though there may be no coating on the tongue, the breath, as well as the exhalations and perspiration, is usually offensive.

The skin and its appendages manifest evidences of perverted nutrition. The skin is dry and scaly, and there are often cutaneous eruptions, eczematous in character. The hair and nails are dry and brittle. Temporary palpitation of the heart is a common symptom, this being functional in character, usually, and is often aggravated or brought on by eating. Slight jaundice may be present, and careful palpation may detect hepatic congestion. Nervous symptoms,

such as headache, giddiness, insomnia, with oppression of breathing at night, requiring an upright position, melancholy and despondency with erratic irritability and anxiety, are prominent features, due rather to digestive disturbance than to the presence of lithic acid. The urine is usually high-colored, and deposits a sediment upon standing until cool. This may consist of uric acid, urea, phosphates, and oxalate of lime. Examination of the heart may detect arterial tension, and such a condition may persist years without apparent serious results, though finally gout, arterio-sclerosis, or contracted kidney will become fully developed.

Diagnosis.—The persistent and distressing gastric disturbance following eating, and the almost constant presence of an excess of uric acid and phosphates in the urine, will distinguish this condition.

Prognosis.—Where treatment is begun early, and intelligently pursued, there is little doubt of success, provided the patient will assist by adhering to a proper diet.

Treatment.—Attention should be paid to the habits of the patient as regards exercise, diet, and the general condition of the alimentary canal. Careful examination should be made to determine, if possible, a local cause for the flatulency and indigestion. Hypertrophy of the spleen will demand the use of *carduus marianus*, *polymnia*, *ceanothus*, or, if there be pronounced malarial complication, *grindelia squarrosa*. Hepatic congestion will suggest *chelidonium*, *nux vomica*, or *nitro-hydrochloric acid*. To relieve the excess of uric acid in the blood, the *citrate* or *carbonate of lithia* should be taken in three- or four-grain doses, dissolved in water, three times daily. *Piperazin* is another remedy for this condition, and a very reliable one. Three or four grains may be taken at a dose, four times daily.

Cabinet vapor baths, aided by the tonic faradic treatment, exert a highly beneficial influence here, promoting cutaneous secretion and invigorating the organs of digestion and circulation, as well as relieving insomnia and painful conditions. The patient should remain in the bath from fifteen to thirty minutes, and it should be repeated two or three times each week. It will assist other measures very much, and should not be omitted.

Careful inspection of the condition of the rectum should be made, and any evidence of disease here be corrected. Rectal pockets and papillæ should be incised, and hemorrhoids, ulcers, and other disease properly treated, the sphincter ani, meantime, being well stretched.

The *diet* should consist largely of milk and vegetable food, butchers' meat being an aggravating cause of the difficulty. Fruits are not objectionable, and may be partaken of freely. Only once a day

should any form of meat be included in the dietary, and this should then be used sparingly. *Quantity* should also be considered, and the amount of food consumed limited to the actual needs of the body.

In order to promote free peristaltic action and a vigorous abdominal circulation, a copious clyster of strong salt water should be used every morning, soon after breakfast, and repeated, if a free evacuation does not follow the first injection; and upon retiring at night and rising in the morning, the whole abdominal surface, especially that over the epigastric region, should be vigorously slapped with the open hands. This may seem a hardship at first, but it will finally become a source of pleasure.

Mineral waters are beneficial, and plenty of pure water is always proper. Alcoholic beverages are not admissible, and tea and coffee should be substituted by a single cup of weak cocoa at breakfast, milk or water being taken at other times.

V. DIABETES MELLITUS.

Synonyms.—Glycosuria; Glucosuria; Glycohæmia; Mellituria.

Definition.—A constitutional disorder arising from malassimilation, characterized by the presence of sugar in the urine.

Etiology.—It has been discovered that an area exists in the medulla oblongata (on the floor of the fourth ventricle) which presides over the glycogenic functions, and that if this part be irritated, either experimentally or accidentally, sugar soon afterward appears in the urine. Concussion of the brain, cerebral hemorrhage, softening of the brain, or other cerebral disease, such as cirrhosis or pressure from tumor, may give rise to glycosuria. Any cause of irritation of the glycogenic center, even mental stress, such as severe mental strain, grief, or sudden shock from fright or surprise, may result in this disease. Alcoholism, pregnancy, indigestion, and the immoderate use of sugar and new wine have been ascribed as causes.

Pathology.—Sugar is found in the blood, and in the secretions and excretions of all organs, though most abundantly in the urine. The amount may equal nine or ten parts in a thousand of the blood, and here are also found glycogen, acetone, and kreatin, the proportion of fat also being greater than normal.

Nearly all the vital organs manifest evidences of degenerative change as the disease progresses. The liver is hyperæmic, with areas of fatty degeneration, and the lungs show points of catarrhal pneumonia with gangrenous tendencies, tuberculous deposits, or patches of pleuritic inflammation. The spleen is enlarged, congested, and hardened. The kidneys are hyperæmic, and bear evidence of

parenchymatous inflammation. The heart is pale, soft, and flabby, and the muscles generally are pale and dry. The brain is variously altered, sometimes being cirrhotic, at others softened, and tumors may be present about the fourth ventricle, suggesting a probable cause of the disease. Wandering parasites (cysticerci) may burrow in the region of the diabetic area and cause the irritation essential to the disease. The pancreas is often notably altered, atrophy and fibroid degeneration being the condition. Emaciation is a marked feature in autopsies, and the skin is usually the seat of various degenerative changes, such as boils, carbuncles, and gangrene.

Symptoms.—Two forms occur, the acute and the chronic. The acute form commonly attacks young persons or those below middle age, while chronic diabetes is most usually observed in those of advanced life. Acute cases are rapid in their course, terminating fatally within a few weeks, while chronic diabetes may linger for years.

In any event, the *onset* is usually insidious. The subject may be impressed that he is losing strength and flesh, that there is unnatural thirst, and that he is obliged to rise frequently to urinate, during the night. Sexual desire soon becomes abolished, and intolerable itching of the genitals or other parts, with unnatural dryness of the skin and mucous membranes, is present. Arrest of the menses occurs in women, often with troublesome pruritus pudendi, as well as itching of the cutaneous surface.

Thirst is a prominent and distressing symptom, and a large amount of water is consumed, the greatest quantity being demanded an hour or two after meals. Digestion is often good in chronic cases, and the appetite may be ravenous.

The *tongue* is dry, red, and glazed, or, in some chronic cases, covered with a dry brown coating; the throat is dry, and the saliva scanty and viscid, or absent. The gums are pale and retracted, and bleed easily, and the teeth soon become carious. The breath and exhalations from the body generally are marked by a sweetish odor. In acute cases there may be nausea and vomiting, with dyspeptic symptoms, and intense headache followed by delirium and coma may occur.

The *special senses* may be involved, especially that of sight, soft cataract or amblyopia sometimes developing. The temperature, pulse-rate, and respiration are usually below normal. *Mental* symptoms are prominent in most cases of diabetes. The patient is irritable, peevish, restless, and melancholic, with periods of dullness and apathy.

The Urine.—The urine is a subject of considerable importance

in this disease, as it is the principal element to be considered in diagnosis. Occasionally there is not marked increase in quantity, but usually the amount voided is enormously large, amounting, in mild cases, to from six or eight pints, to as high as fifty in extreme cases, within twenty-four hours. The specific gravity is high, ranging from 1.025 to 1.045. The urine is pale and clear, almost as limpid as water, and possesses a sweetish odor and taste, and an acid reaction. Tests for sugar detect a varying quantity (from one to ten per cent) of sugar present. Ten or twenty ounces, and even as much as one or two pounds, may be excreted in twenty-four hours.

Various tests, to determine the presence of sugar in suspected urine, have been recommended. A few of the most important ones are appended:

Trommer's Test.—Add a few drops of a dilute solution of sulphate of copper to a drachm of urine in a test-tube, and then an equal bulk of liquor potassæ. Boil, and if sugar be present, a yellow or orange red precipitate occurs.

Fehling's Test.—Add a drachm of Fehling's solution to a test-tube and boil. If the solution remain clear, add a few drops of the suspected urine, and boil again. If there be sugar present, the yellow suboxide of copper is precipitated.

Fermentation Test.—Add a particle of yeast to a test-tube full of urine, and invert the tube so it will stand in the same liquid, in an open vessel. If there be sugar present, fermentation will go on with the formation of carbon dioxide, which accumulates in the upper portion of the tube and gradually expels the urine.

Bismuth Test.—To half a drachm of the suspected urine add an equal bulk of solution of potassa and a pinch of subnitrate of bismuth, and boil for one or two minutes. If sugar be present, black, metallic bismuth deposits.

Complications.—Various complications arise as results of the continued presence of the saccharine material in the blood:

Cutaneous affections, such as eczema, boils, and carbuncles, and sometimes gangrene, are apt to arise. Pruritus may attend any of these, or arise independently. The frequent calls to urinate and the local irritation caused by the affected urine give rise to severe erythematous inflammation about the genitals in some cases, especially in women, and balanitis is not uncommon in men. In other cases there may be only troublesome itching.

The *urinary tract* generally, may be involved. Cystitis or nephritis may be present, and albuminuria may develop in connection with arterio-sclerosis.

Pulmonary complications are frequent, pneumonia or pulmonary

gangrene arising, the pleura sometimes participating. Tuberculous complication often occurs in the bronchi or lung parenchyma, in which the tubercle bacilli are present, demonstrating true tuberculosis.

A wide range of *nervous symptoms* attend this disease. Diabetic *coma* is a frequent complication among young subjects. It may sometimes be the first symptom to be noticed, and it terminates many cases suddenly. Headache, delirium, and dyspnœa, with subsequent cyanosis, rapidly failing pulse, exhaustion, and coma, with death in four or five days, are the usual symptoms. Sometimes sudden exhaustion and coma come on after severe exertion, the patient succumbing in a few hours. In other cases, the patient may be suddenly attacked with severe headache and intoxication, without previous dyspnœa or exertion, and rapidly sink into unconsciousness and fatal stupor.

Disturbances of *motion* and *sensation* arise in various instances. *Peripheral neuritis*, characterized by numbness, tingling, or neuralgic pains darting through the lower extremities, sometimes occurs, attended by loss of muscular power, with absence of knee-jerk, power in the extension of the feet, and even loss of strength in the arms and legs.

Diagnosis.—The loss of flesh and strength, with thirst and marked increase in the amount of urine voided, will suggest the disease, and urinary analysis will settle the question.

Prognosis.—Mild cases of glycosuria may recover under treatment, and chronic cases may be modified, for years, but there can be little hope held out in acute attacks.

Treatment.—Though modification of the diet cannot be expected to exert a curative influence, it doubtless lessens the severity of the disease, and thus aids in its successful management. Mental strain is aggravating in its influence, and the patient should be removed from all causes of worry or mental effort, and be allowed to live a quiet and even life, in a mild climate, where winter and summer nearly meet. As the capillary circulation is usually poor, the underclothing should be of flannel or silk, and the skin should be kept moist and open by a daily cabinet vapor bath (unless the patient be too much exhausted) and by daily fatty inunction with massage.

The *diet* should consist of easily digested meats, fish, poultry, and game without reserve, except liver, crabs, lobsters, and oysters; liquids, except those containing sugar, such as beer, sweet wines, and sweet aërated drinks; and vegetables, except potatoes, turnips, parsnips, squashes, vegetable marrow, asparagus, corn, beets, and artichokes. Fruits should be prohibited, except lemons, oranges, and

currants. The bread should be restricted to gluten and bran bread, and almond and cocoanut biscuits. All wheat and rye bread should be avoided, while such farinaceous foods as rice, hominy, tapioca, semolina, sago, arrowroot, and vermicella are not permissible. It is said that gluten flour obtained in this country contains too great a proportion of starch—that that from Paris and London contains a much smaller amount of this element, and is to be preferred. As the breads especially prepared for diabetics are all unpalatable, and soon become distasteful to the patient, it may be better to allow a restricted quantity (a few ounces) of ordinary bread daily, though this should then be well toasted, to disorganize the sugar and dextrin.

The milk diet advised by Donkin and at one time popular in diabetes has failed to prove generally satisfactory, though some cases improve on it.

As a substitute for sugar, saccharin and glycerine may be employed.

The *medical treatment* of diabetes is not yet very successful, though we have improved upon older methods. *Syzygium*, when a recent preparation can be obtained, removes the sugar from the urine in many instances. Unfortunately, there is no guarantee of the character of an article obtained, and it has often been so long in the market as to have lost its therapeutic value. Three or four grains of the powdered seeds, in capsules, constitute a dose, to be repeated three or four times daily. In the use of any remedy in this disease, perseverence is a necessary virtue. *Nitrate of uranium* controls profuse urinary discharge, and thus modifies many unpleasant features. Where there is much pain and restlessness, the following capsule may answer a good purpose: ℞ Phenacetin gr. ii, nitrate of uranium 3x trit., gr. iii. M. This may be given every two or three hours.

Rhus aromatica and *lycopus virginicus* have both, according to written reports, accomplished cures of diabetes. Whether these were cases of genuine glycosuria or merely diabetes insipidus, where agents controlling an excessive hydruria are expected to succeed, still remains to be satisfactorily proven. There is no reason either of these remedies should fail. Lycopus is invigorating to the digestion, promotes normal activity of the heart and arteries, and allays gastric and enteric irritability, thus seeming well adapted to some of the conditions of this disease, while it controls relaxation of the renal capillaries, thus being commendable for trial, at least. It has the reputation of having cured many cases of diabetes mellitus. It should be given in ten-drop doses of the specific medicine, every three or four hours. Professor I. J. M. Goss, in his Practice of

Medicine, describes the case of an old man (74 or 75 years of age) whose urine showed a specific gravity of 1.045 and upon evaporation yielded a considerable quantity of what was apparently saccharine material, in which rhus aromatica effected a complete cure within three months. Lycopus and uranium were used the first month of treatment, with only the result of lessening the volume of water passed. The following month he was put upon 30-drop doses of rhus aromatica three times daily, and, at its expiration, there was a noticeable diminution in the amount of urine voided, and its specific gravity was 1.032. The same treatment was continued for another month, the patient meantime gaining flesh and strength rapidly, and, after the third month he reported himself well, and so continued. It is to be regretted that a chemical analysis was not made at the beginning and ending of treatment in this instance, that more positive statements might have been made.

Opium possesses the reputation of limiting the progress of the disease, codeia being the form generally preferred, as it is less constipating. The drug may be begun in small doses—half a grain three times daily—and gradually increased, as the patient becomes tolerant, to six or eight grains in twenty-four hours.

Dr. J. G. Pierce has employed *bromide of potassium* in the treatment of cases where the disease was brought on by injury, such as falls resulting in concussion, with promising results. In one of his cases, that of a young girl, it was brought on by extreme grief at the death of her mother, and here a complete cure resulted.

During coma little can be done, though inhalations of oxygen and intravenous injections of a three-per-cent solution of bicarbonate of sodium have been recommended and employed, but not with very satisfactory results.

VI. DIABETES INSIPIDUS.

Synonyms.—Polyuria; Polydipsia.

Definition.—A constitutional disease, characterized by extreme thirst and the excretion of a large amount of colorless urine of low specific gravity, containing neither sugar nor albumin.

Etiology.—The etiology of this disease is obscure, but many circumstances point to a nervous origin. For instance, it is apt to follow blows on the head, or injuries to the occipital region of the skull. Bernard discovered a spot in the floor of the fourth ventricle in animals, the puncture of which was followed by this condition. It occurs most commonly in young persons, and heredity seems to exert an influence. Excesses in drinking, both of ice-water and

alcoholic liquor, have been followed by it. It sometimes appears during the course of such visceral lesions as hepatic cirrhosis and abdominal tumors, some impression being made here upon the renal nerves, in all probability. Cerebral tumors have caused it, and sunstroke, apoplexy, and other brain lesions have been followed by it. Males are more subject to it than females, probably because they are more liable to causes of violence which predispose to it.

Pathology.—Various degenerative changes have been found in the central and sympathetic ganglia. Anatomical lesions of the kidneys and bladder sometimes occur, the bladder being hypertrophied, and the pelves of the kidneys, and ureters dilated. Chronic pulmonary complications may arise, with fatal termination by tuberculosis.

Symptoms.—The principal symptoms in the beginning are inordinate flow of urine, and thirst. The urine is limpid, colorless, of low specific gravity, and shows no reaction with agents employed for testing for sugar. The disease may come on insidiously or suddenly, the amount of urine voided finally reaching from thirty to sixty pints per day. The specific gravity varies from 1.003 to 1.008. The reaction is faintly acid, there is a greenish, opalescent color, and uric acid, urea, and kreatin are present in larger than normal quantities. As the flow of urine increases, the thirst becomes proportionately pronounced, and the amount of liquids consumed bears a direct relation to the quantity of urine voided.

As the disease progresses the skin becomes dry and harsh, the nails brittle, and the temperature subnormal. The general condition of the patient varies considerably in different cases. Sometimes the excessive thirst and profuse urinary flow are about all the symptoms noticed, the subject maintaining tolerably good health otherwise. In other cases digestive derangements, with loss of appetite and gastro-intestinal disturbance with prostration and emaciation, gradually advance. Sometimes vomiting and rapid emaciation attend, followed by cough, hectic, and fully developed phthisis. Salivation is an occasional symptom, and it may persist throughout.

Diagnosis.—Polyuria may arise in other diseases besides diabetes. Hysterical persons sometimes void large quantities of urine in a short time, but the polyuria is of short duration, at erratic intervals. In diabetes insipidus the profuse discharge is constant, and observation of a patient for a month will settle the question as to the diabetic nature of the disease. Absence of sugar will distinguish it from diabetes mellitus.

Prognosis.—The disease may continue for years, without seriously undermining the health. Spontaneous recovery some-

times occurs during the course of acute diseases, and death, when a fatal issue attends, is usually the result of intercurrent affections. Spontaneous recovery is rare.

Treatment.—Whenever the cause can be ascertained, it should be removed. The skin should be warmly clothed in flannels, and a warm and equable climate should be chosen for residence.

The *medical* treatment will consist of those agents which tend to constringe the renal capillaries, either by direct action, or through the vasomotor nerves. *Rhus aromatica, lycopus, nitrate of uranium, jaborandi,* and other agents, have been used with varying success, to control the excessive urinary flow. As the disease is probably nervous in origin and probably often reflex, the difficulty of directing the specific agent to the point of irritation is apparent; and, even if the trouble arise from lesions about the medulla, we are at a loss to prescribe a remedy which will maintain a steady and permanent control over it. *Glycerole of gallic acid,* in half-teaspoonful doses, sometimes acts beneficially in restricting the excessive discharge of urine. A solution of twenty grains of quinine in an ounce of tincture of muriate of iron, dose, ten drops every three hours, sometimes serves a good purpose. *Phosphoric acid* bears an excellent reputation, and is reported to have accomplished numerous cures. *Scilla maritima,* in fractional-drop doses, sometimes exerts an excellent influence. Full doses of valerian, ergot, antipyrine, and various other drugs, have their advocates.

An active state of the skin is advantageous, and this may be brought about by the use of the *cabinet vapor bath,* repeated two or three times weekly. The galvanic current, one pole at the nape of the neck and the other at the loins may be tried, but its efficacy is doubtful, even though it is highly recommended.

VII. RICKETS.

Synonyms.—Rhachitis; Rachitis.

Historical Note.—The term rickets is supposed to be either from the Saxon word "rick," a hump, or from a Dorsetshire verb "rucket," to breathe laboriously. The disease was first described by English writers, and the first case noticed appeared in Dorsetshire. Therefore many writers retain the original term, "rickets," to designate the disease. Those of more classical turn prefer the term, "rhachitis," which is derived from a Greek word signifying spine.

Definition.—A constitutional disease affecting children, characterized by disturbance of normal processes of ossification, attended by enlargement of the epiphyses, with softening of the bones and resulting deformity.

Etiology.—Three periods of life are especially liable to this disease, viz., the fœtal period, the infantile period, and that of adolescence, malnutrition being responsible. It is said that large numbers of still-born children are found, upon careful investigation, to be rickety. The usual period for the development of the disease is between the sixth month and the third year. Both sexes are equally susceptible, statistics showing about an equal number to be affected. Heredity plays an important part, though constitutional weakness is all that can be claimed in this respect. Protracted lactation and repeated pregnancies lower the vitality of mothers, the younger children of large families being more prone to rickets than the older ones, excessive and prolonged lactation resulting, doubtless, in deterioration of the mother's milk. As European families are much larger, as a rule, these facts may suggest a reason for the greater prevalence of the affection in Europe than in America, where it is comparatively rare except among children of European immigrants, and negroes. Poor ventilation, dampness, and want of sunlight are believed to predispose to it, especially when infants are weaned early and fed upon farinaceous diet, starchy food tending to the formation of lactic acid. The disease is more prevalent in large cities than in rural districts or small towns.

As to the actual condition which is responsible for the improper bony development, there are numerous theories, all taking for granted that there is a lack of phosphate of lime to supply the developing bones with the proper amount of earthy material somewhere. The lactic-acid theory assumes that there is an excess of lactic acid generated in the alimentary canal by imperfectly digested starchy material, and that this removes the lime destined for the bones from the blood in the form of soluble salts, and irritates the bones at the same time. Others have asserted that insufficiency of earthy salts in the food gives rise to it, but this is disputed, as rickets may occur under the best of conditions of this kind. Lack of fats and proteids in the diet of rhachitic children has been supposed to be responsible for it. Syphilis, malaria, bronchitis, and other conditions have been held responsible for the development of rickets, but it must still be admitted that there is some obscure element at work in most cases which cannot be accounted for, and that the specific cause is yet to be determined. The rickets of adolescence is probably a result of an infantile attack which has been barely warded off, or not fully recovered from, but which has remained in a latent state throughout childhood, and developed through the important systemic changes then occurring.

Pathology.—During the active stage the most marked pathological changes occur at the points of junction between the epiphyses and shafts of the long bones. The cartilage which separates these parts is normally thin (about two millimeters in thickness), but in rickets it becomes expanded into a thick, reddish-gray, translucent cushion, while the adjacent bony structure is enlarged and softened. The vascular layer which underlies the periosteum is softened, pulpy, and thickened, the periosteum itself being thickened and swollen, and its attachment to the bone more than ordinarily tenacious; and a pale-red, pulpy fluid infiltrates the epiphyses, periosteum, and bones. The bones of the skull, the ribs, and the wrists are most frequently involved, the proportion of inorganic material being very much decreased, it being supposed that lack of phosphate of lime in the system results in the absorption of that element from bones already ossified to supply growing bones in the developing child. The bones become soft and yielding, bodily weight and muscular action tending to twist them out of their normal shape. The liver and spleen take on various pathological changes. The spleen becomes engorged and enlarged, and the liver is sometimes affected with fatty infiltration. The lymphatic glands are occasionally enlarged.

When the process of ossification begins to be reëstablished, the bone is laid down so rapidly that layers of new formation appear on the surface, causing, in many cases, increased deformity.

Symptoms.—Rickets is usually the outcome of a protracted period of ill health, in which there are no obviously specific symptoms. The child will be noticed to be pale, restless at night, with a disposition to kick the bedclothing off, and there is usually a marked tendency to relaxed sweats, especially about the head. It gradually grows pot-bellied, the tissues becoming flabby and inelastic, and most cases manifest a general tenderness about the body, the child crying out with pain when handled. This symptom may be so marked that the gentlest effort at moving the patient may provoke intense pain, as manifested by shrieks from the sufferer. The "paralysis of rickets" may now become developed, the child losing the ability to walk, if it has already learned to do so, and it may lose the use of the arms as well. However, there is no real nervous lesion in such a case, the inability to use the parts depending upon muscular weakness and tenderness of the bones and periosteum instead of lack of nervous impulse. This condition is termed "Parrot's disease," and precedes the marked changes in the bones, and therefore is liable to be confounded with latent meningeal or spinal trouble.

Sometimes there is the complication of bronchitis with these indications, and this may give rise to elevation of temperature and other febrile symptoms. In other cases the chronic disease may develop without intercurrent complication, and little active constitutional disturbance be manifested. Thus, acute and chronic rickets have been described, though there is essentially no difference between them, except as the incidental complication may determine the condition.

After a somewhat protracted period the *osseous changes* begin to appear. The ribs and the wrists manifest the earliest and most marked changes. The points of junction between the ribs and costal cartilages protrude as a "rosary" of bead-like enlargements, readily felt upon palpation, and the wrists assume characteristic shapes, enlargement of the lower extremeties of the radius and ulna imparting a noticeable bulging to the parts. The typical head of rickets is now gradually developed. The forehead becomes high, square, and prow-shaped, with decided prominence of the frontal eminences; the parietal eminences may also be prominent, and the skull is elongated. The intellectual powers are not necessarily retarded by rickets, the brain usually developing, and expanding its functions as though there was no disease present, though there may be exceptions to the rule, and dementia, idiocy, or imbecility be the condition. The face of a rhachitic child is large above and diminutive below, as the jaws are usually small and the lower one retracted, giving the chin a retreating appearance. This affords an intelligent expression to the countenance, the child impressing the observer as an individual of precocity, though marks of ill health are portrayed by the enlarged and superficial veins of the scalp and forehead, and open anterior fontanelle. The sutures of the skull close more slowly than usually, and a gutter may be left along their course, following ossification. Dentition is also delayed, the first tooth appearing about the ninth month, and the last deciduous tooth about the third year. When developed, the teeth may present the characteristic appearance described as "Hutchinson's teeth."

Thinning or wasting of the tables of the skull may occur, until portions of its surface become so attenuated as to yield to gentle pressure, imparting the sensation to the finger of the crackling of parchment. This is termed "cranio-tabes." It is not often observed in this country.

Hyperæmia of the brain and meninges is liable to attend, and *hydrocephalus* is extremely apt to follow such a condition.

Deformities of the chest are very liable to attend rickets, and sometimes a condition of this character is all that may be found to

attest the presence of the disease, or its results. As the framework of the thorax becomes softened, the muscles and atmospheric pressure tend to bend the bones out of shape, the most yielding point being at the costo-sternal junction. Sometimes the thorax is flattened laterally, the sternum being projected forward, constituting "pigeon breast," or pectus carinatum. At other times the deformity may be unilateral, one side yielding more than the other, or one side being depressed and the other bulging. The diaphragm exerts a tension on the ribs which is sometimes marked in rickets, a line of depression, corresponding to the points of its attachment, encircling the thorax. In some cases the results of the distended abdomen may remain after the bones have become hardened and the abdomen has flattened, the lower ribs remaining rolled outward and upward, maintaining a peculiar deformity.

Spinal curvature is common in this disease, three forms being described, viz., kyphosis (backward curvature), scoliosis (lateral curvature), and lordosis (forward curvature).

Softening of the long bones may result in bowing of the forearm, bow-legs, knock-knees, etc. The sacrum may yield to the pressure from above and throw the direction of the pelvic axis backward, the condition imparting a squatting posture to the person when standing. Almost every deformity imaginable may arise in this disease, from distortion of the bones.

Diagnosis.—The disease should be suspected when a child becomes pallid, with doughy, flabby tissues, and tendency to profuse perspiration about the head habitually, especially at night. Such symptoms are sufficient for therapeutic diagnosis at least, and they sound a warning which should not be unheeded, for now is the time to administer the treatment to forestall serious osseous changes. After these have begun a short time, there can be no mistaking the condition, the deformities and general symptoms combining to make the picture complete.

Prognosis.—Permanent dwarfing, added with various deformities, follows the subsidence of the disease. Many of the lesser deformities, such as enlargement of the epiphyses, diminish with growth, though spinal curvature, pigeon breast, and rhachitic skull mark the results of the disease throughout life. During its course, various complications tend to fatal results, the principal of these being bronchitis, broncho-pneumonia, diarrhœa, hydrocephalus, and amyloid degeneration of internal organs. Proper treatment, begun early, usually benefits in a short time.

Treatment.—The most important consideration in treatment is attention to hygienic methods. If the child be nursing, and the

mother seem to be in indifferent health, it must be removed from the mother's breast and a healthy wet-nurse substituted. Or, if this be impossible, it should be put upon properly prepared cow's milk, the various infant foods containing too much sugar and dextrine for such children. If the cow's milk be sweetened, sugar of milk and not cane-sugar should be used. The child should be kept in the open air much of the time, as oxygen and sunlight are important aids in treatment.

One of the most efficacious medicines is *calcarea carb.*, 3x trituration, administered in two- or three-grain doses, four times daily. This is especially indicated when there are nocturnal head-sweats, and it should be prescribed immediately upon the appearance of this symptom, as it may prove prophylactic against further development of the disease.

Silica 3x is another remedy indicated in sweating about the head, and it exerts an excellent influence over reparation of bony and cartilaginous structures. It may be alternated with calcarea carb. or employed alone, with good results.

Schuessler's tissue remedy is worthy of trial in rickets, as it often proves efficacious. *Calcium phosphate* will usually act better in minute doses in this disease than in the large ones often advised, and the 3x trituration, in two- or three-grain doses, three or four times daily, is worthy of confidence.

Phosphoric acid, in minute doses, phosphorus, and cod-liver oil are other remedies which have been highly recommended.

The *tonic faradic* treatment will be found to assist the action of medicines very much in the management of this disease. It may be repeated two or three times a week.

VIII. SCURVY.

Synonym.—Scorbutus.

Definition.—A chronic constitutional disease, due to deficiency of fresh vegetable and animal diet, characterized by anæmia, prostration, sponginess of the gums, and tendency to hemorrhage.

Etiology.—Scurvy, in times past, was preëminently a disease of the sea, the crews of slow sailing vessels, who had exhausted their vegetable food and lived on salt pork and biscuits for a long period of time, being the ones principally affected. In these times, when facilities for preserving vegetables in hermetically sealed cans for an indefinite period has become perfected, the disease is comparatively rare. Armies, in time of war, are sometimes obliged to subsist on salt meat and hardtack for a protracted period without fresh food,

and the men are then liable to contract scurvy. During recent times, the miners of Alaska have been the most common sufferers, deprivation of fresh vegetables and other ingredients supplied by them being very common to that country during the winter months. Several cases of the kind have been at the Maclean Hospital within the past two years, all Alaskan miners recently landed from the north.

Considerable difference of opinion exists as to the identity of the specific causal factor. Some follow Garrod and believe that absence of the potassic salts is answerable for the pathological developments, while others believe that the condition arises from the lack of malates, citrates and lactates, from which the carbonates, which render the blood alkaline, are derived. At any rate, a gradually diminishing alkalinity of the blood attends, and there seem to be good grounds for logical reasoning from cause to effect in this connection.

Physical influences, outside of that of diet, and mental states undoubtedly exert a certain effect in the production of the disease. Homesickness, especially when attended by other depressing influences seems to lessen the resisting power of the system; epidemics of the disease on convict ships in olden times, and in prisons, where the diet would hardly warrant it, go far toward establishing the proposition that mental influence of a depressing nature is an important causal factor.

All ages are liable to it, though elderly persons are most susceptible. Starvation alone seems not to dispose to it, as scurvy has never followed the most prolonged fast; only those who eat food lacking the proper elements being attacked.

Pathology.—There is decreased alkalinity of the blood, which is dark, fluid, and does not coagulate readily. Deficiency of the potash salts has been demonstrated. The capillaries present evidences of alteration of the endothelial cells and are choked, in places, with red corpuscles. Ecchymosis is common, the skin and subcutaneous tissue, the muscles, the joints, the subperiosteal tissue, the mucous and serous membranes and the internal organs all being more or less involved. Hemorrhages occur in the internal organs, especially in the kidneys and bladder. The gums are especially involved, being swollen, spongy and hemorrhagic, and often ulcerated, even so that the teeth become loosened or fall out. Parenchymatous changes occur in the spleen, liver, kidneys, and heart. The spleen may be markedly enlarged and swollen.

Symptoms.—The disease advances insidiously. Gradual loss of flesh, with prostration and pallor, attract first attention. Sponginess of the gums may now be noticed, these parts being swollen, tender, hemorrhagic, and fungous in appearance. Loosening of the teeth commonly occurs, though the affection of the gums is not always

present. The tongue is swollen and livid, ecchymosis may appear in the mucous membrane of the mouth, and the breath is fœtid and offensive. Sometimes the salivary glands are swollen. Ecchymoses may now be observed about various parts of the cutaneous surface; these are first seen about the legs, then on the trunk and arms, especially about the hair-follicles. These may be minute, purple spots, or may be larger, and may cause cutaneous swelling. The face appears bruised and swollen, presenting a livid appearance. The skin is dry, rough and generally of a muddy pallor, though it may be sallow and leaden in hue, and slight blows or bruises are followed by extensive extravasations.

Severe darting pains affect the limbs, especially about the calves and popliteal spaces, and node-like swellings, from deeply seated ecchymoses, often appear on the shins.

The circulation is feeble, the pulse small and slow, except when there is excitement, and there is palpitation of the heart, with anæmic murmurs, and dyspnœa upon slight exertion. Where the disease is advanced, syncope may follow even moderate exertion. Sleeplessness, disordered vision, and other nervous disturbances are common.

The bowels are constipated, and the urine scanty, often albuminous, and there is diminution of the normal ingredients, except phosphoric acid and the potash salts.

Diagnosis.—The history of the case and a careful inspection of the gums will distinguish between scurvy of these parts and mercurial poisoning. In purpura there are not the marked lesions of the gums that usually attend scurvy, and they occur in isolated cases, while scurvy is liable to appear in epidemics. Purpura also resists the restorative influence of lime-juice, while this agent readily relieves scurvy.

Prognosis.—If the conditions which give rise to the disease are removed, and it is not far advanced, the prognosis is good. Death results in from ten to fifteen per cent of cases, gradual heart-failure, meningeal hemorrhage, extravasation into serous cavities, intestinal inflammation, and other intercurrent conditions usually carrying the patient off. When complicated with syphilis or chronic alcoholism, the prognosis is less favorable.

Treatment.—When fresh vegetables are not to be had during long intervals, scurvy is to be feared, and, if possible, prophylaxis should be observed. Hall and Kane asserted that the eating of raw meat acted as a preventative of scurvy during their Arctic experience, while cooked meat would not. Raw potatoes have been used for the same purpose by the miners of Alaska, and with good effect, according to reliable reports. The daily consumption of a small amount of lime- or lemon-juice serves the best purpose, probably, though limes

and lemons are not always to be had. Other antiscorbutics are mustard, radishes, cabbage and water-cress.

When a patient is seriously sick with scurvy, he should have perfect rest, as the great debility of the heart and other vital organs forbids that he should exert himself in the least. He should remain in bed, and take three or four ounces of lime-juice or lemon-juice, well diluted in water, every day. On account of the tenderness of the gums, the food should be liquid in form, and should consist of beef tea, meat soups, broths, soups thickened with vegetables, milk, and eggs. Return to solid food should be gradual, and the use of lemon- or lime-juice should be continued for a prolonged period during convalescence.

The *medicinal* treatment should consist of a fifteen-drop dose of a reliable preparation of *berberis aquifolium*, administered in a little water, and repeated four or five times daily.

Infantile Scurvy (Barlow's Disease).

Imperfect adaptation of food may give rise to scurvy in children, as well as in adults. It is frequently the case that infants and young children are deprived of fresh vegetables and their immediate derivatives until a scrobutic condition is engendered. An infant at its mother's breast, or fed upon fresh cow's milk, though not taking vegetables, derives, from this source, constituents immediately elaborated from them, and the nourishment is properly adapted to the demands of nature. But when artificial foods are employed, or the child is fed upon condensed milk (which has been cooked), and no fresh milk or other fresh food is employed, scorbutic conditions are liable to arise, in the midst of plenty.

The *symptoms* resemble those of scurvy in adults, though, as the disease will not occur as an epidemic, the physician may overlook the true state of affairs. The skin presents a muddy pallor, the gums are spongy, and a purpuric rash appears on the lower extremities, and later bruise-like ecchymoses will be noticed upon various parts of the cutaneous surface. Officious practitioners may attempt to cure such cases by lancing the gums, with the mistaken idea that they are swollen from efforts at dentition, and provoke fatal hemorrhage. In one case of this kind which came under my observation, the child continued to bleed from the incision for two weeks, and finally died apparently from loss of blood. There is probably pain and tenderness in the calves and other parts of the lower extremities, as the child cries when they are moved or put upon the stretch. Obscure swellings occur upon various parts of the lower extremities, due, probably, to extravasations under the periosteum; and these are usu-

ally symmetrical—appear upon both extremities consecutively, in about the same location. The limbs are drawn up at first, but later become relaxed and lie immobile and flaccid, as though paralyzed, with the toes turned outward.

The anæmia is profound, and the patient is extremely prostrated and asthenic. The eyelids are puffy, one or the other or both are ecchymosed, and protosis or falling of the eyeballs may occur, consecutively, due, doubtless, to ecchymoses in the orbits. There may be slight elevation of temperature, but the pulse is feeble and irregular. The general aspect of the child will be suggestive of rickets, though the symptoms will be much more accute than in that disease.

Treatment.—Fresh milk should at once be substituted for artificial foods. If the infant is very young the best substitute for prepared foods will be a wet nurse. However, it is not always possible to obtain such a substitute, and fresh cow's milk, properly diluted, and sweetened with milk sugar, will be in order. It has been noticed that raw meat has been found a preventative of scurvy, while cooked meat is not; and so it seems to be with cooked milk—and condensed milk possesses the objection of having been cooked.

Further than this the treatment will be similar to that for adults affected with the same disease Lemon- or lime-juice, well diluted, should be used sparingly, and meat-juice or gravy, with seived potato, potato-soup, and other digestible forms of vegetable diet, commensurate with the age and condition of the child. should be allowed.

IX. PURPURA.

Purpura is a disease liable to attend a variety of pathological conditions. Strictly speaking, it is the term applied to extravasations into the skin from systemic causes apart from those of scurvy.

Symptomatic purpura may arise as a concomitant of some other disease, or from the action of drugs or poisons. It may arise from malignant endocarditis, pyæmia, septicæmia, typhus fever, measles or small-pox. The rashes which attend the exanthemata are examples of purpuric eruptions. Again, it may be *toxic*, and due to poisoning from venomous reptiles; the action of certain drugs, such as bromide of potassium, iodide of potassium, copaiba, quinine, and some others. A not uncommon form of symptomatic purpura is that which arises as a complication of *arthritic* disease, and this may, to all intents and purposes, be a severe attack of inflammatory rheumatism attended by a purpuric rash, covering the legs, and even the body and arms. In other instances, the rheumatic symptoms may not be so marked, and may amount only to muscular pains. A form of rheumatic purpura, described as peliosis rheumatica, or Schönlein's disease, where the purpuric symptoms are extreme, amounting to œdema of the skin, with

various eruptive characters, such as wheals, vesicles, etc., complicated with multiple arthritis, sore throat, and elevation of temperature, to 101°—102° F., sometimes occurs.

Cachetic purpura may arise during the progress of cancer, tuberculosis, Hodgkin's disease, albuminuria, or during senility.

Neurotic purpura may appear during the course of certain nervous affections attended by organic changes in a given area of nerve supply. Locomotor ataxia, acute myelitis, and severe neuralgias are instances where such purpuric conditions have arisen.

Mechanical purpura may attend venous stasis of any form, and may occur after severe vomiting, paroxysms of whooping cough, or seizures of epilepsy. In these cases the purpuric spots will be most likely to appear in the face.

Henoch's purpura usually occurs in children, and is another variety of symptomatic purpura. Various portions of the body may be affected, and the disease may continue for years, with occasional outbreaks between periods of freedom. The lesions may occur in the skin, in the intestinal mucous membrane, in the joints or in the kidneys. The cutaneous symptoms may consist of erythematous eruptions, instead of simple purpura. The intestinal lesions may be manifested by crises of pain, vomiting and diarrhœa; the kidney disturbances by attacks of hemorrhagic nephritis, the arthritic complications by pain and swelling in the joints.

Purpura Hemorrhagica.

True purpura is recognized by pronounced purpuric spots or ecchymoses, with hemorrhages from the mucous membranes. It is otherwise known as *morbus maculosus Werlhofi*, and is attended by changes in the blood-vessels, or in the blood itself, probably both combined, and extravasations into the connective-tissue spaces of the rete mucosum, and into the mucous membranes. The serum soon absorbs from the skin, leaving the red corpuscles, which may either undergo gradual absorption or degenerate, leaving a permanent pigmentation. The extravasated blood in the mucous membranes is liable to escape, and hemorrhages from the mouth, nose and other mucous surfaces may attend. Extravasations into the serous membranes sometimes, though rarely, occur, and the peritonœum, pericardium, pleuræ and pia mater may be the seat of purpuric spots. The muscles, bones, periosteum, conjunctiva and retina occasionally suffer from purpuric extravasations.

Symptoms.—Malaise and digestive derangements may precede the onset of the disease for several days or weeks. The eruption appears suddenly, coming out on the extremities and trunk first and

usually stopping there, though the head and face may also be affected. Bleeding from mucous surfaces may now set in, and profound anæmia may rapidly develop from epistaxis, hæmatemesis, or hæmoptysis. Loss of blood may result fatally, or cerebral hemorrhage may carry the patient off. Sometimes the disease assumes marked malignancy, and terminates fatally within twenty-four hours, with large purpuric extravasations in the skin. Cutaneous hemorrhages and extreme prostration are the leading symptoms, bleeding from the mucous membranes being absent or death occurring before it begins. This is termed *purpura fulminans*. Recovery is gradual, the purpuric spots disappearing, in favorable cases, in ten days or two weeks.

The *diagnosis* is to be made between this disease and scurvy, and will readily be made in adults, who have been necessarily deprived of fresh vegetables; though in children more care is required. In scurvy swelling and ulceration of the gums is a prominent symptom, while it is liable to be absent in purpura. It will hardly be confounded with malignant forms of eruptive fevers, where epidemic tendencies and a high temperature are readily recognized.

Treatment.—Symptomatic purpura should be managed according to the special condition giving rise to it. The exanthemata should be recognized and properly treated; and rheumatic purpura will yield to treatment for ordinary rheumatism. A study of conditions, and proper treatment for special demands, must be the duty of the attending practitioner. In the treatment of purpura hemorrhagica such remedies as *berberis aquifolium*, *cistus canadensis*, *arctium lappa*, *corydalis*, etc., should be thought of. Where hemorrhages are severe and threatening, *erigeron canadensis*, may be of service. In malignant forms, such agents as *echinacea*, *lachesis*, or *baptisia*, may be of avail. Sometimes ten drops of tincture of *muriate of iron* every three or four hours answer a temporary purpose, though ferruginous preparations are not usually to be depended upon. Ordinary hemostatics may fail utterly to control purpuric hemorrhage, though it may be well to try them.

X. SCROFULA.

Definition.—Scrofula is a term applied to many different conditions of the system, depending upon a peculiar diathesis now believed to be tuberculous. On this account most medical authors ignore the term, and consider scrofula as a form of tuberculosis.

Etiology.—The scrofulous diathesis is usually inherited, and may be due to syphilitic, intemperate or phthisical progenitors. The children of parents closely related by blood are liable to inherit a scrofulous tendency; and it may be acquired during early life, through the influences of bad air, food and other antihygienic surroundings.

Symptoms.—If scrofula and tuberculosis are not identical, they are so closely related as to be interchangeable. However, subjects may survive scrofulous inflammation and live a lifetime afterward, where tuberculosis in ordinary form would soon prove fatal. Extreme *chronicity*, with tendency to caseous degeneration, are its leading features. It is principally a disease of children, and is manifested by transparency of the skin, blue veins, lustrous eyes, precocity of intellect and nervous irritability. Scrofulous subjects are either markedly of the encephalic temperament, or else are of the lymphatic type, in which case they have large heads, coarse features and thick, flabby skins, with overproduction of fat about the nose and upper lip. Glandular enlargements are early characteristics of scrofulous children, though prior to these developments cutaneous inflammations, of chronic character, are liable to appear, especially about the corners of the mouth, upon the edges of the eyelids or in the ears. Tonsillar enlargements are common, and catarrhal affections are difficult to cure and tend to return upon slight provocation. Pharyngeal, laryngeal and bronchial catarrh are induced by slight causes, and in little girls vaginal leucorrhœa and troublesome vulvitis are not uncommon. Slight injury to the joints is liable to result in suppurative inflammation of destructive character, and caries of the joints, with tuberculous deposits, often develops without the aid of traumatism.

Diagnosis.—There is little possibility of mistaking scrofulous inflammation for any other disease. The diathesis bears its evidence with it, and the chronicity of the affection aids in determining its character. Scrofulous deposits contain tubercle bacilli, and these are also found in inflamed scrofulous glands.

Prognosis.—Scrofulous children often pass through childhood to adult life and live to a fair old age, though crippling from joint affections is not rare. Tuberculous intestinal disease, acute hydrocephalus, croup and pulmonary diseases are very liable to arise in such children.

Treatment.—Prophylaxis is to be considered. Blood relations should avoid marriage among themselves; broken down, phthisical and syphilitic persons should not marry at all.

A plain, nutritious diet, with plenty of open-air exercise in sunshine, should be encouraged. Cutaneous eruptions should be met with *calcium sulphide*, *calcarea phos.* and *berberis aquifolium* or *stillingia sylvatica*. Affections of the joints are best met by radical surgical measures. Affections of the lymphatic glands yield best to *calcium sulph.* or *calcium fluoride*, though such vegetable remedies as *coryalis*, *phytolaccca*, *stillingia*, *berberis aquifolium* and other reputed alteratives are not to be neglected here.

XI. HÆMOPHILIA.

Definition.—A constitutional fault, of hereditary character, consisting of a tendency to uncontrollable bleeding upon slight provocation, and even spontaneously in many instances.

Etiology.—Hæmophilia is a systemic fault which, in the majority of instances, is transmitted from mother to son. The daughters of such a mother are not liable to be bleeders, but the male children they bear will probably be subject to hæmophilia. Thus the weakness is handed down to the male portion of the family, while the tendency to transmit the weakness is entailed upon the female portion. While the sons are bleeders, their children seem to be exempt, the disease being transmitted through the female alone. This rule has its exceptions, and there is undoubtedly occasionally a female subject who proves to be a bleeder. Therefore, it has been estimated on good authority that about one in thirteen of the subjects of hæmophilia is of the female sex. There are exceptions to the rule that hæmophilia is hereditary, as it is occasionally acquired; though just the essentials to its origin from healthy stock is not known.

Pathology.—There are no peculiarities of structure about the blood-vessels of subjects of hæmophilia usually, though in some instances anatomical changes have been found in the capillaries. Unusual thinness is the only peculiarity liable to then attract attention. Probably lack of tonicity is more at fault than tenuity of structure, as proper tone about the stomata in the minute vessels would be important in the control of capillary hemorrhage. Hemorrhages have been found in and about the joints, and inflammation of the syovial surfaces. Possibly the morbid state may depend upon some peculiar fluidity of the blood, rather than upon fault of the bloodvessels.

Symptoms.—Uncontrollable bleeding from trivial causes is the leading feature of the disease. A slight scratch, blow or cut, the extraction of a tooth, or even epistaxis, may result in prolonged and alarming hemorrhage, which persistently resists all ordinary means of relief.

Sometimes the bleeding is traumatic and sometimes spontaneous. Traumatic bleeding may be interstitial, and may consist of petechiæ and ecchymoses, as well as bleeding into the joints. Spontaneous bleeding may occur from the nose, mouth, stomach, bowels, urethra and other internal organs, as well as from the skin, at various points, such as the navel, vulva, scrotum, eyelids, ears, finger tips, etc.

The bleeding is a capillary oozing, but it may be so profuse as to occasion rapid dripping of blood from the part and cause speedy prostration. Continuing on, day after day, it soon becomes alarming, and may finally result in fatal syncope. When the bleeding is

into the joints, there is pain and swelling not unlike the symptoms of rheumatism, especially if it is accompanied by elevation of temperature and accelerated pulse.

Diagnosis.—Where a knowledge of the family history can be had, the diagnosis will be much simplified. Prolonged bleeding from trivial causes will hardly occur, except in purpura, and the symptoms of this disease are not likely to be mistaken.

Prognosis.—When hæmophilic manifestations appear very early in life, the outlook is less favorable than when they are deferred until adult life. More than fifty per cent of boys who become bleeders very young, in a given number of cases, die before the seventh year. It is believed that the longer a bleeder survives the greater chance he has of outgrowing the tendency. In female patients subject to hæmophilia the menstrual and parturient functions are fraught with more than ordinary danger.

Treatment.—Exciting causes should be avoided as much as possible. Boys who belong to hæmophilic families should avoid active habits, so that danger from traumatism may be lessened. The avocations of such persons should be selected with this object in view. One of the very worst cases of this kind I ever treated was that of a carpenter, who scratched the back of his hand slightly with a saw. I had previously, several years before, treated him for dangerous bleeding following the extraction of a tooth. Tooth-extraction and all minor surgical operations should be avoided in such patients, as far as possible.

When bleeding has begun, absolute rest should be required, and *oil of erigeron* should be administered, in ten-drop doses, repeated every hour or half-hour. If this fails, small doses of *carbo. veg.* may be tried. *Rhus aromatica* is a good remedy, though the oil of erigeron has proven the best remedy for me. *Ergot, gallic acid, tannin* and a score or less of other commonly known hæmostatics might be suggested, to be tried in their turn, as there is no known specific. *Subsulphate of iron*, locally, is the best aid to internal measures. A low diet is to be commended.

SECTION IV.

DISEASES OF THE DIGESTIVE ORGANS.

I. DISEASES OF THE MOUTH.

Herpes Labialis.

This affection is quite a common one, and seldom requires attention, as it is self-limiting, in the majority of cases. It often appears with a cold, and a common name for the condition is "cold-sores." Herpes labialis often appears during the course of cerebro-spinal fever, as well as in the course of other fevers, and requires no special attention. When it becomes chronic, or persists for a longer time than usual, *phytolacca* may be used internally, while a dilution of *grindelia robusta* is applied locally. ℞ S.m. or normal tinct. phytolacca, gtt. x-xx, aqua ad. ʒiv. Dose, a teaspoonful every two hours. ℞ Saturated tinct. grindelia robusta ʒi, aqua ad ℥i. Apply every three or four hours. In long-standing cases, *berberis aquifolium* may act better internally than phytolacca.

Simple Stomatitis.

This is the commonest form of inflammation of the mouth, and all ages are subject to it. It usually results from the action of irritants, such as hot or highly-seasoned food, strong drinks or tobacco. In children, dentition or gastro-intestinal irritation may account for it. It often arises during the acute specific fevers.

There is redness and dryness of the mucous membrane at first, involving a greater or less portion of the oral mucous membrane, with excess of secretion later on. Burning of the surface attends, with smarting upon attempts at mastication. Sometimes the tongue is swollen and furred. There may be slight elevation of the temperature, especially in children, although constitutional symptoms are not marked.

The treatment will consist of a weak dilution of glycozone, ʒi to boiled water ℥ii, or ʒi of grindelia robusta to water ℥iv, used as a wash. A solution of chlorate of potassium ʒi to water ℥iv often answers well. Internally, ℞ Phytolacca ʒss, aqua ad ℥iv. Dose, a teaspoonful every hour. The local applications will usually be sufficient.

Aphthous Stomatitis.

Synynoms.—Follicular Stomatitis; Croupous Stomatitis.

Definition.—An ulcerative form of stomatitis involving the mucous follicles of the oral mucous membrane.

Etiology.—Aphthæ may attend any inflammatory disease of the tongue or mouth, though age and hygienic surroundings may exert an influence. It most commonly occurs among children, and may even appear as an epidemic. It frequently attends the acute infectious diseases, one of the worst epidemics I ever saw being a complication of chicken-pox. It is quite common among children as a sporadic and idiopathic affection, and may be caused from indigestible food remaining in the mouth, unripe fruit, candy, etc. Cachetic conditions and bad hygienic surroundings may be blameable for its appearance. Some women are troubled with aphthous ulcers at each menstrual period, and pregnant and nursing women are sometimes affected by a stubborn form.

Pathology.—Semi-transparent vesicular elevations appear on the mucous surfaces of the cheeks, gums, and tongue and around each of these is a reddened base; these constitute what are termed "aphthæ." Sometimes they are very numerous, studding the mucous membrane thickly, and at other times they are few and scattering. As these rupture they leave irregular ulcers, which heal slowly. If several coalesce they form a single, large, irregular ulcer, which may be tardy about healing. Sometimes there may be deeper sloughing, and the submucous tissues are excavated.

Symptoms.—When occurring in nursing infants, the first sign will probably be a refusal to take the nipple; and if the child attempts to nurse it will quit often and cry peevishly, because of the pain excited. With older persons mastication is painful and difficult, and the taking of fruits or anything sour excites excruciating pain; and the same is true of hot food or drink. Slight febrile excitement may be present, the submaxillary glands may be hardened and swollen, and ptyalism may be more or less of a factor. Children are liable to suffer from a diarrhœa, as a complication.

Treatment.—The practice of treating these ulcers by the applision of caustics, or even by local washes, is usually unsatisfactory, though a weak dilution of *hydrozone* sometimes assists, when used as a wash. The specific treatment consists of the use of *phytolacca*, which selectively influences the oral mucous membrane with reparative effect. As there is usually more or less febrile complication, the addition of aconite to the prescription is advisable. ℞ Specific phytolacca gtt. x-xxx, specific aconite gtt. i-vi, aqua ad. ʒiv. Administer a teaspoonful every hour. Where ptyalism is a prominent and

persistent feature and there is marked disposition for the vesicles to coalesce and form large, ragged ulcers, *jaborandi* is an important remedy. Ten or fifteen drops of the specific medicine (or any other reliable form) should be added to the prescription just offered. Nursing sore mouth is very stubborn, and will seldom yield to such treatment. Attenuations of *lachesis* have had the best influence here of any remedy I have used.

Fœtid Stomatitis.

Synonyms.—Ulcerative Stomatitis; Putrid Sore Mouth.

Definition.—An ulcerative form of stomatitis, which occurs in crowded communities, like jails, camps, etc., where surroundings are detrimental to health. It may arise from the use of a community drinking cup or similar cause; and a predisposition to it is encouraged by unwholesome food, bad ventilation, carious teeth or those upon which there is an accumulation of tartar. Bacteriologists believe that the specific etiological factor consists of a microbe; and the belief has been entertained, in some quarters, that the disease is identical with the foot-and-mouth disease which infects cattle and that it is conveyed in the milk. Other theories have been advanced, but no positive knowledge exists as to the real specific causal factor. It sometimes occurs sporadically.

Symptoms.—The ulcerative process begins at the margins of the gums and spreads along over two or more aveoli. The gums and surrounding mucous membrane become swollen and spongy, bleed easily, and ragged ulcers form along the gingival margin, with tendency to rapid increase in size. The breath is foul, the tongue is coated. There is severe aching and throbbing pain in the mouth, which is intensified and becomes burning and stinging upon taking food. There is ptyalism, usually profuse, and the submaxillary glands are swollen and tender. Constitutional symptoms develop; there are dryness of the skin, elevation of temperature, sleeplessness and emaciation.

Treatment.—*Hydrozone* is the best remedy for use here, though constitutional treatment may be required to assist its action. The ulcers should be treated locally with it, either by spraying the gums or by applying it with a swab, and the excavations along the fangs of the teeth should be deeply syringed with it by the aid of a hypodermic syringe. In addition, a wash of it should be used in the mouth, for general cleansing purposes. The drug may be diluted with one or two parts of water, though at first full strength may be used, for cleansing deep excavations. In connection with this, ten or fifteen drops of a reliable preparation of *echinacea* should be administered, for its systemic effect, and repeated every hour or two.

Berberis aquifolium is a remedy which should not be forgotten in these cases. *Chlorate of potassium* has its ardent admirers, though I am of the opinion that it has been overrated. I have never tried the local influence of *grindelia robusta* here, but believe, from its effects in other forms of ulceration, that it would prove highly beneficial in fœtid stomatitis.

The *diet* should be liquid in form, and unirritating. Milk, raw eggs, custards and other semi-solid and easily-digested foods may be be taken during convalesence.

Mercurial Stomatitis.

Definition.—An inflammation of the mouth due to the specific influence of mercury upon the tissues.

Etiology.—Mercurial ptyalism is a common affection with the patients of many allopathic physicians, The idea that mercury should be pushed until the gums are "touched" is yet in favor with many of them, and with those who do not believe that ptyalism is essential belief in the curative effects of mercury in many conditions is a common one. Consequently, mercurial poisoning frequently occurs, and occasionally an Eclectic is called upon to administer relief in such cases.

Symptoms.—The patient may complain of a metallic taste and profuse dribbling of saliva. Upon inspection the gums will be found swollen, reddened, dusky and sensitive upon mastication. The breath is offensive, and ptyalism soon becomes a disgusting annoyance to the patient. As the disease continues the tongue becomes swollen, the submaxillary glands enlarged and tender, and the gums may ulcerate along their gingival margins. Caries of the aveoli, with frequently recurring gum-boils and premature decay of the teeth, is likely to be a remote result.

Treatment.—Minute doses of *jaborandi* offer as good results as any remedy. Add from ten to fifteen drops of specific jaborandi to half a glass of water and order a teaspoonful every two or three hours. *Phytolacca* is also an excellent remedy, though not as reliable as jaborandi. Add twenty drops of the saturated tincture to half a glass of water and order a teaspoonful every three hours. *Chlorate of potassium*, used as a wash, is in great favor with some, and it is claimed that it exerts an antidotal influence against mercury. Diluted *hydrozone* should not be forgotten, and if other remedies fail try *grindelia robusta*. ℞ Specific robusta ʒii, aqua ʒiv. Gargle frequently.

The *diet* should be bland and unirritating, and of such form as to be easily masticated. In bad cases the diet should be liquid in form, and free from acids and high seasoning.

Eczema of the Tongue.

Synonyms.—Map Tongue; Geographical Tongue.

Definition.—A desquamation of the epithelium of the tongue, which occurs in circinate patches, imparting to the surface of the organ a map-like appearance.

Etiology.—The etiology is obscure, ordinary causes of eczema of the skin being at the foundation of the trouble. It occurs most frequently in children, though adults are subject to it. Some regard it as a gouty manifestation, though this is not probable. Indigestion may attend it, and possibly the condition may be due to gastric disturbances.

Symptoms.—The patchy tongue presents a striking appearance, and attention is thus often called to it. Sometimes there is itching and heat upon the affected surface, but mental perturbation, from fear of more serious developments, is the unpleasant feature of many cases.

Treatment.—Thus far treatment for this affection has afforded little satisfaction. The continued use of *berberis aquifolium* may relieve the burning, when this is present, and even restore the normal appearance. *Graphites* has relieved one case which I have observed, though it has proven futile in others. *Calc. phos.* 3x is worthy of a trial. Where specific remedies fail to relieve, attention to the general health will be commendable. Such cases are chronic, and naturally recuperate slowly.

Parasitic Stomatitis.

Synonyms.—Thrush; Muguet; Soor.

Definition.—A fungous disease of the mucous membrane of the mouth.

Etiology.—Thrush is a fungous growth which develops from transplantation of the oidium albicans, a yeast-like fungus, which affects children most commonly, but to which adults, when greatly debilitated, may become subject.

Pathology.—The development of the oidium albicans and its parasitic companion studs the mucous membrane of the mouth with patches of pultaceous, creamy masses, which may coalesce until the surface of the tongue and buccal cavities are largely covered with it. In children the palate is a favorite place for its lodgment. The epithelium of the mucous membrane becomes loosened, secretion is arrested, and the part becomes dry and dusky. The oidium albicans consists of spores and filaments resembling the yeast plant in certain respects.

Symptoms.—The mouth becomes hot and sensitive. There is dryness of the mucous membrane, though the action of the salivary glands may be increased, with acidity of the secretion. The mucous membrane becomes swollen, the lips everted. Patches of thrush increase in size, thus forming a membrane which can be scraped off, leaving the mucous surface more or less excoriated and sensitive. The buccal mucous membrane, the lips, the roof of the mouth and even the fauces and tonsils may become affected, the whitish incrustation covering more or less of their surface with ragged patches. Sometimes the disease extends to the œsophagus, and even the stomach and bowels may be invaded and troublesome diarrhœa, with flatulence and green stools, attend.

A microscopical examination will settle disputes in *diagnosis*, the distinctive features of the oidium albicans being thus determined.

Treatment.—Careful attention must be paid to the diet, especially in cases of children fed upon the bottle. Everything should be kept carefully cleansed—bottles, tubes and nipples—and no sour or fermented food should be allowed. Sometimes a radical change in the character of the food used will be imperative. The substitution of a wet nurse may sometimes be necessary. Antiseptic mouth-washes must be assiduously employed, and diluted *listerine*, weak *lime-water*, or weak solutions of *bicarbonate of sodium* may be used. In adults, weak solutions of equal parts of *carbolic acid* and *glycerine* or diluted *hydrozone* may be needed, to cleanse the mouth frequently.

Constitutional treatment is important, special remedies adapted to particular cases being called for. Hygienic surroundings are desirable, and where many children have been crowded together isolation in healthy localities is desirable. Care should be observed in the use of spoons, nursing-bottles and other feeding implements, that the contagium be not conveyed to healthy subjects. *Aconite* and *phytolacca* will be appropriate to relieve the irritation of the mucous membrane.

Gangrenous Stomatitis.

Synonyms.—Cancrum Oris; Noma.

Definition.—An affection occurring in children, characterized by rapid and progressive gangrene of the side of the face, having for its starting point the gums or cheek.

Etiology.—This hideous malady has for its principal factor the effects of mercury. Allopathic authorities fail to state this fact in their treatises on medical practice, but I have never known it to occur except in children who have been previously mercurialized. The prostration following acute fevers may favor its development and

ravages, as may also unsanitary conditions, but the irritating and debilitating influence of mercury on the tissues involved in the start undoubtedly gives rise to it.

Symptoms.—The mucous membrane of the gums or cheek of the affected side is first attacked by ulceration of phagedenic character, a deep, sloughy ulcer in the part being the first symptom noticed. As this rapidly spreads, the adjacent skin and underlying tissues become indurated and purplish, and the sloughing continues to extend until the cheek is perforated. Sometimes the entire cheek melts away, the ulceration extending to the chin and tongue, and even the eyelids and ears may be involved.

Marked constitutional symptoms are developed as the case progresses. The temperature rises to 103° to 104° F., the pulse becomes accelerated, and grows feeble later on, nausea and diarrhœa supervene and profound prostration and death finally follow. In other cases the sloughing gradually ceases, leaving the patient with a ghastly, grinning, skeleton-like aspect upon one side, the teeth and gums being exposed as far back as the angle of the lower jaw. Other cases may be arrested before the destructive action has progressed so far.

Treatment.—*Echinacea*, both locally and internally, is the best agent with which I have had extended experience in phagedenic ulceration. In such a case as this the system should be well saturated with it, and it should be applied to the part constantly. Ten drops may be administered every hour, and a twenty-five per cent dilution in water should be kept in contact with the part, on compresses. *Hydrozone* spray is another excellent local remedy.

From the very favorable reports received of the use of preserved bovine blood *(bovinine)* in phagedenic ulceration, I would expect it to benefit here. It might be used locally, either on antiseptic gauze or injected into the affected tissues, while the proper dose was employed internally. It might be used in connection with echinacea (or Lloyd's echafolta). Grindelia robusta ʒi to aqua ʒii might be found a useful adjunct, if applied locally.

Pyorrhœa Alveolaris.

Definition.—This term strictly signifies suppuration of the alveoli, but is here limited to a peculiar kind of inflammation of the alveoli and surrounding soft structures characterized by the deposit of a dark, slate-colored material on the roots of the teeth, with rather wide-spread inflammation of an insidious character, the teeth becoming loosened and the gums destroyed, without much pain or well-marked sensitiveness. It is distinct from the disease caused by the deposition of calcareous material about the fangs of the teeth (tar-

tar) and distinct from alveolar abscess, which is usually confined to the space lying at the extremity of a single fang.

Etiology.—The exciting cause of the disease is the slate-colored deposit, the origin of which is still in doubt. It has been ascribed to serumal deposit of a gouty nature, from the fact that uric acid has sometimes been found in it, but this is insisted on as being accidental by competent persons, and the theory of a gouty origin now seems to be pretty well disposed of in the negative. The practice of administering mercury to salivation doubtless has something to do with a loss of vitality about the affected parts, which predisposes them to afford lodgment to the incrustation, and the impaction of particles of food may cause thrombus of the pericementum, with subsequent transformation of the arrested blood into earthy material. It seems to involve the entire thickness of the pericementum, and is firmly attached to the cementum (which invests the dentine of the fang).

Pathology.—The concretion usually scales off the cementum readily about the shaft of the fang, but at its point or extremity the cementum is roughened, and the removal of the morbid accumulation requires the assistance of chemicals, in addition to instrumental means. The presence of the foreign body gives rise to irritation of the gingival margin, and the gum gradually shrinks away from the fang, leaving it exposed and finally revealing the dark incrustation. The soft tissues in the sockets become gradually involved, and the bone is attacked later, concealed pockets of pus forming about the fangs in the aveoli, until the teeth become loosened, the gingival margins soft and spongy and the soft structures about the apices of the fangs honeycombed by burrowing suppurative action. The teeth may finally drop out, the patient suffering so little pain as to scarcely realize that they are being destroyed.

Symptoms.—The disease is insidious. Slight reddening of the edges of the gums will be the first symptom noticed, and if these are now slightly retracted the slate-colored deposits will be found just below the gingival margin, out of sight of superficial inspection. These are small at first, but they gradually involve more or less of the entire surface of the fang, spreading destruction to the structures of the sockets as they advance, though there is little pain or tenderness to attract attention. Sponginess of the gums and loosening of the teeth are later developments, and finally the teeth fall out from destruction of their attachment. If a probe be passed down along the sides of the teeth, cavities will be found between the fangs and alveoli and extending into the gums.

Treatment.—The best plan to pursue is to refer the patient to a competent dentist. However, there may be circumstances where it will not be practical for the patient to reach a dental office, and

the physician, in such cases, should know how to manage the affection.

Destruction of the pus and cleansing and stimulating of the cavities should be a prominent part of the treatment The removal of the incrustation is imperative, in order that a cure may follow. *Hydrozone* should be injected into all the cavities by means of a hypodermic syringe, the needle of which may be converted into a blunt tube by removal of the point. The hydrozone may be diluted by the addition of an equal part of boiled water at first, if it causes pain in full strength, and it should be warmed before use, as cold solutions are unpleasant to the sensitive structures. Removal of the incrustation must be accomplished with minute scrapers or chisels, obtainable at any dental depot. The scraper is curved near the point and the fang is raked from the point toward the crown, whlle the chisel is straight and the motion is from the crown toward the extremity of the fang. Repeated operations should be practiced in order to accomplish the complete removal of the accumulation, a delicacy of touch thus being attained which will enable the operator to detect the presence of a particle of the accumulation when it cannot be seen. The repeated use of hydrozone will gradually loosen masses which are at first firmly adhered, and every treatment with it should be followed with a search over each affected fang for incrustations. Finally each operation should be concluded by the insertion of a mixture of equal parts of fluid extract of *quercus alba* and *oil of cinnamon* into every cavity and along the fang of every affected tooth, the application being made by dipping a small chisel into the mixture before each probing.

Success in treatment depends on perseverance and attention to details. Several weeks of the use of hydrozone and search for incrustations are necessary, the applications being made every other day at least. As treatment progresses the swelling of the gums subsides. the teeth become more firmly fixed and tenderness disappears. The teeth should be inspected at intervals, however, for a long time afterward, in order to avoid a return of the disease.

II. DISEASES OF THE SALIVARY GLANDS.

Hypersecretion of the Salivary Glands.

Synonym.—Ptyalism.

Etiology.—Ptyalism occurs under a number of conditions. One of the most common causes is the abuse of mercury, weeks of excessive salivary action following some cases of unfortunate mercurialization. Pregnancy is another condition where excessive action of the

salivary glands may prove a source of annoyance. Some vegetable agents provoke ptyalism, jaborandi, muscarin and tobacco being notable examples, though their effects are usually transient. Ptyalism may occur during the course of some acute fevers, though the opposite is the rule. Small-pox is occasionally attended by it.

Symptoms.—The symptoms are unmistakable. The mouth, however frequently emptied, continues to fill with saliva, which is thin and watery, dribbles upon the chin and is thus a source of continual vexation. Irritation of the mouth and lips often attends, and speech is interfered with by the provoking presence of excessive fluid in the buccal cavity.

Treatment.—The successful treatment of the ptyalism from mercury is often attended by a great deal of difficulty. The salivary glands frequently seem so debilitated and relaxed that ordinary remedies for pytalism fail to produce much effect. Small doses of *jaborandi*, repeated frequently, sometimes succeed. ℞ Specific jaborandi gtt. x. aqua ʒiv. M. Sig. A teaspoonful every two hours. *Hydrastis* does well here, though it will often fail. The *galvanic current* sometimes succeeds rapidly, and is usually very successful in the ptyalism of pregnancy. Take an ordinary tongue depressor and, laying it on the tongue of the patient, bring in contact with the other part (if it be metal) the metal terminal of a conducting cord connected with the negative pole of a galvanic battery, the patient holding, meantime, a wetted sponge attached to the positive pole in one of her hands. The current should be about four or five milliamperes in strength (about eight or ten four-ounce zinc-carbon cells, in good order). After holding it there a few seconds remove it, to allow the patient to rest the tongue, then repeat once or twice afterward in the same order. Repeat this treatment every other day for a week or more. Sometimes the positive to the tongue will be more satisfactory. *Faradism*, used in the same manner, sometimes succeeds, though it is not as positive as galvanism.

Arrest of the Salivary Secretion.

Synonym.—Xerostoma.

Etiology.—This is supposed to be due to disturbance of the function of the salivary nerve center. A majority of the cases which have occurred have been in female subjects and accompanied by nervous phenomena. The disease is very rare.

Symptoms.—The symptoms are purely local, the general health rarely being disturbed. There is remarkable dryness of the tongue, mucous membrane of the cheek and palate. The tongue is parched and dry, sometimes cracked, and the remaining mucous membrane

of the mouth is smooth, dry and shining. Mastication and deglutition are attended with difficulty, the mouth becoming clogged by remnants of food adhering to the gums.

Treatment.—The only successful treatment that leaves permanent results is by the use of *galvanism* as described under the treatment of ptyalism. Here the negative pole should invariably be applied to the tongue. *Jaborandi* may temporarily promote the action of the salivary glands, though here it is given in ten-drop doses of the specific medicine, or in still larger quantities.

Inflammation of the Salivary Glands.

This may occur under different circumstances and require consideration from various standpoints. *Specific parotitis* has already been discussed among the specific infectious diseases. *Symptomatic parotitis* occurs from a variety of influences. It may arise during the infectious fevers, either from continuity of oral inflammation along the salivary duct or from septic inflammation through the blood. Suppuration usually attends an inflammation of this character, active inflammatory action following. Another condition in which symptomatic parotitis may ensue is that following facial paralysis with peripheral neuritis.

Injuries or disease of the abdomen, pelvis, kidneys or genital organs are sometimes attended by parotitis, also such diseases as ulceration of the stomach and such injuries as blows upon the testicles, the introduction of a pessary, a surgical operation on these parts or other pelvic, genital or abdominal organs. In these cases the etiology is not well defined, though probably the causes are septic in nature.

In the *treatment* of such cases small doses of *jaborandi* or *potassium chloride* 3x, in connection with a mild current of *galvanism* or *faradism*, may bring about resolution and avert suppuration. When this becomes inevitable, warm poultices should be employed until evidences of suppuration are present, and early incision for the evacuation of pus should then be practiced.

III. DISEASES OF THE PHARYNX.

Acute Pharyngitis.

Definition.—An acute inflammation of the mucous membrane of the pharynx and adjacent surfaces.

Etiology.—This is a catarrhal condition, due to sudden changes which give rise to colds, the pharynx being a favorite place for the location of the irritation in those who are apt to clear the throat

often by hawking and empty swallowing, when there exists a slight irritation there. In some instances the disease may occur as an epidemic, though here, probably, there is some specific cause at work, and the condition a form of specific infectious disease rather than a purely local inflammation. In the exanthematous fevers, such as small-pox, scarlatina, rubeola, etc., an exanthematous inflammation of the pharynx attends as a part of the febrile condition. *Erysipelatous* inflammation of the pharynx may originate as an extension of facial erysipelas through the auditory meatus or nasal duct, or it may arise independently, from direct erysipelatous infection of the part.

Symptoms.—Pain in the pharynx, with a disagreeable sensation of dryness, irritation, fullness and difficulty of swallowing, mark the outset of the disease, these symptoms coming on a few hours after exposure to draughts or dampness. The patient frequently attempts to clear the throat by hawking and swallowing. The voice is muffled and there is a short, dry cough. There may be slight febrile disturbance, especially in children. The extent and severity of the inflammatory action is best determined by inspection of the throat. Sometimes the inflammatory blush (redness) extends forward, involving the palate and pillars of the fauces. The posterior nares are often involved, the patient complaining of burning there and making frequent efforts to clear the passages of screatus. Headache is now a frequent symptom, this continuing for several days until the acute symptoms have subsided. The uvula is often involved and it may be œdematous and elongated.

Acute pharygitis is occasionally *erysipelatous* in character, and then there is a peculiar bright redness to the affected part, the inflammation extending rapidly and widely, œdema and puffing of the inflamed area being marked and constitutional symptoms severe.

Treatment.—Ordinary cases of acute pharyngitis recover rapidly on the following prescription: ℞ Green-root tinct. of phytolacca ʒi, Lloyd's or other reliable fluid extract of aconite gtt. v–x, water ʒiv. Mix, and order a teaspoonful every hour. Where the inflammation is severe and stubborn, the addition of ʒi of *jaborandi* to the preceding prescription will be of much service, and often much advantage will attend the use of a gargle of one part of *echinacea* to four or five of water, its use being repeated frequently—every half- or quarter-hour. In erysipelatous pharyngitis the internal use of echinacea is essential, two or three drachms of the specific medicine being added to half a glass of water and a teaspoonful ordered every hour. Jaborandi does well here in combination with the echinacea, in about the quantity already indicated. Erysipelatous pharyngitis with puffiness of the tissue especially indicates minute doses of *apis*.

When the vault of the pharynx and the posterior nares are severely affected, the following prescription may answer better than the treatment first suggested: ℞ Specific apocynum cannabium ʒi, aconite gtt. v–x, water, ʒiv. M. Sig. Take a spoonful every hour. In other cases of this kind *sambucus canadensis* may be used in place of the apocynum, in the same quantity.

Cold water packs are the best local application in this affection, though some prefer heating fomentations. A small towel may be wrung out of cold water and folded to the appropriate size, and the throat bandaged with it, the application being renewed every three or four hours.

In connection with the treatment the patient should be enjoined to avoid empty swallowing, hawking and attempts at forcible removal of screatus from the posterior nares.

Should the uvula become elongated and œdematous during the course of acute pharyngitis it should not be excised at that time, as the operation would be liable to aggravate the local difficulty and serious results might happen, especially in erysipelatous pharyngitis.

Phlegmonous Pharyngitis.

Retro-pharyngeal abscess, attended by severe inflammatory action, occasionally occurs. It may be due to local injury, such as the irritating influence of hot food or penetration by spiculæ of bone, though it usually depends upon caries of the cervical vertebræ. The inflammatory action may be treated by *phytolaccca* and *aconite*, as in simple acute pharyngitis, pus being evacuated early. *Silica* 3x should then be thought of.

Gangrenous Pharyngitis.

This may occur in connection with diphtheria, small-pox or other infectious disease. In addition to the gangrenous local condition, there are usually typhoid symptoms and profound prostration, with, in many cases, fatal results. *Echinacea*, *lachesis*, *baptisia* and other remedies of their class should be thought of early. Frequent spraying with diluted echinacea may accompany its internal use.

Chronic Pharyngitis.

Synynoms.—Pharyngeal Catarrh; Clergyman's Sore Throat; Pharyngitis Sicca.

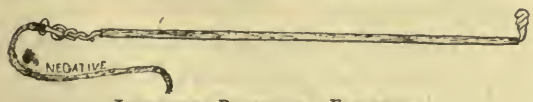
Etiology.—This condition may be developed from repeated attacks of acute pharyngitis, or it may rise imperceptibly from subacute inflammation of the part. It is common in public speakers, auctioneers and hucksters, who overstrain the voice. Excessive

smokers and drinkers are also specially liable to suffer with it. It may be brought on by persistent efforts to clear the throat by empty swallowing, hawking, removal of screatus from the posterior nares, and such causes.

Pathology.—The mucous membrane of the naso-pharynx and posterior wall of the pharynx are relaxed, the venules are dilated and the mucous glands are each surrounded by proliferation of lymph-tissue. When this is very abundant, the functions of the glands are destroyed and the mucous membrane becomes dry and glistening, constituting *pharyngitis sicca.*

Symptoms.—The patient may not manifest much discomfort, though often there is dryness of the throat on awaking in the morning, with sensation as of a foreign body in the part, provoking hawking and empty swallowing. The mucous membrane of the posterior pharyngeal wall is usually dusky, the veins are enlarged and in follicular pharyngitis there are raised points of bright, reddened tissue distributed upon its surface. A mass of tenacious mucus will usually be found adhering to the posterior pharyngeal wall, extending downward from behind the soft palate. If destruction of the mucous follicles has been accomplished the surrounding mucous membrane will be found dry and shining. A short, dry cough indicates more or less irritation of the larynx. In many cases the uvula is congested and elongated and becomes an additional cause of laryngeal irritation, cough being a common symptom,

Treatment.—The treatment of the catarrhal form of chronic pharyngitis is neither difficult nor tedious. *Galvanism*, applied to the affected part, two or three times a week for a month or more, produces excellent results, if not a complete cure. Confirmation of cure is but a question of time; persevere in the treatment and the cure is sure to follow, if the patient gives the part rest from hawking and unnecessary empty swallowing. An electrode may be improvised with the aid of a section of copper wire, a piece of rubber tubing and a pledget of absorbent cotton, which will answer the purpose admirably. A loop is bent upon one end of the wire, for purpose of connection, the tubing is then drawn on so as to leave about three-fourths of an inch of the further extremity exposed, and this is turned up at right angles, for the attachment of the absorbent cotton, which, when wetted in plain water, constitutes an excellent applicator. When in use, the loop is to be placed and held in contact with the tip of a conducting cord attached to a galvanic battery, while the patient holds a moistened sponge connected with the opposite pole in one hand (after the cot-



Improvised Pharyngeal Electrode.

ton-carrier has been placed in position in the throat). A current of three or four milliamperes (six or eight four-ounce carbon-zinc cells in active condition) should be used in this case. The patient may not be able to endure the electrode in the throat for more than a few seconds at a time as it is liable to produce retching, but the application should be made two or three times at each sitting and repeated twice a week. The hooked portion of the applicator should be carried up behind the soft palate so as to bring the current in contact with that part, where the trouble is almost always most severe. Sometimes the operator may advantageously apply the wetted sponge to the angle of the jaw on each side, that the current may be sent directly through the wall of the pharynx.

The pole to be applied must be selected with reference to the condition of the part, though the negative will usually be most effective. However, if there is great relaxation with profuse catarrhal discharge or if there is marked irritability, the positive may answer better.

In connection with this treatment, considerable benefit may be derived from *collinsonia*, *penthorum sedoides* or *cistus canadensis*. Ten drops of specific medicine of either agent may be administered at a dose in a little water before meals and at bedtime. Such agents, assisted by sprays and gargles of different kinds, may alone accomplish cures, though results are not ordinarily satisfactory.

In follicular pharyngitis and in other forms where hypertrophic spots are apparent, *galvano-cautery* may be used to destroy them, appropriate medicines being taken, meanwhile, to promote a healthy condition of the mucous membrane.

In pharyngitis sicca *jaborandi* possesses specific properties. Ten drops of the specific medicine should be used at a dose persistently four or five times daily for months, attention being paid to the constitutional condition of the patient as well. *Guaiacum* also possesses considerable virtue here, in minute doses.

If the uvula is elongated or relaxed it should be caught with a rat-tooth catch forceps at the tip, drawn slightly forward and half or more of its length snipped off with scissors curved on the flat. This will ensure the permanent removal of one source of irritation.

Arduous use of the voice should be discontinued; smokers affected with the disease should abandon the habit, and those addicted to alcoholism should abstain from all alcoholic liquors.

The habit of wearing a *cold wet pack* on the throat at night is an excellent one.

Ulceration of the Pharynx.

Etiology.—Ulcers of the pharynx may depend upon a number of causes. Among them may be named: (1) Those which arise from

follicular pharyngitis; (2) those from syphilis; (3) those from cancer; (4) those from tuberculosis.

Symptoms.—*Follicular ulceration* is easily detected. The raised, isolated points on the posterior pharyngeal wall, one or more of them being the seat of superficial ulceration without induration or tendency to deep, destructive action, distinguish them from other forms. They should be treated with *galvano-cautery*, or fuming *nitric acid* applied with a pine stick.

Syphilitic ulceration of the throat is more chronic and less painful, the ulcers of secondary syphilis being multiple, while those of tertiary syphilis are deeper and usually single, denoting the erosion of a gumma. The history of the case will assist the practitioner to a clear diagnosis where syphilitic ulceration is present. *Galvanism* is the proper agent for the successful treatment of such cases. The negative pole is applied to the ulcer or ulcers twice a week, the patient holding a positive sponge-electrode in the hand. To assist the local agent the following prescription will be found an important aid: ℞ Specific corydalis ʒss, specific berberis (or Parke, Davis & Co.'s fluid extract) ʒi, simple elixir ad ʒiv. Sig. Take a teaspoonful four or five times daily. When the ulcers are tertiary in character, the addition of iodide of potassium to this prescription may occasionally be desirable.

It may be difficult to always distinguish *cancerous ulceration* from that of syphilis, though it is to be remembered that the ulceration of cancer is usually extremely painful, while that of syphilis is comparatively painless. Cancerous ulceration, moreover, is steadily progressive, while syphilitic ulceration, after developing, is usually stationary. The history of the case will afford additional light upon the subject. The best that can be advised for cancerous ulceration of the pharynx is treatment to relieve the pain. Opiates are of little value, and the only remedy of much use is *echinacea*, which usually renders the condition a comparatively painless one, while the patient retains consciousness and the use of his mental faculties to the last. From ten to twenty drops of the specific medicine may be administered at a dose in a little water every three or four hours during the day. The local application of *grindelia robusta*, repeated often, is to be highly commended here. ℞ Grindelia robusta ʒii to aqua ʒiv. It may be applied with a spray apparatus.

Tuberculous ulceration occurs in some cases of advanced phthisis. The ulcers are ragged and irregular, with ill-defined edges and yellowish-gray bottoms, and are intensely painful. The general condition of the patient and the history of the case will suffice for a diagnosis. The ulcers should be frequently sprayed with *bovinine*, and the same agent should be administered internally in appropriate doses.

An excellent application to this as well as to all other pharyngeal ulcers will be found in *hydrozone*.

Ludwig's Angina.

Synonyms.—Angina Ludovici; Cellulitis of the Neck.

Definition.—A cellular inflammation of the tissues of the neck encountered during medical practice as a complication of specific infectious diseases, especially diphtheria and scarlatina.

Etiology.—In addition to the infection of streptococci, which originates it in the specific fevers, it may arise as a result of traumatism and may even occur idiopathically. In every case it is even possibly due, eventually, to infection from streptococci.

Symptoms.—Swelling begins about the submaxillary gland of one side at first, but it soon becomes general, septicæmia, œdema, glottidis, secondary pneumonia or gangrene of the affected parts proving fatal in many cases in a short time. Termination by abscess is the most desirable end to be expected.

Treatment.—*Echinacea*, *lachesis* and *baptisia*, combined with prompt surgical interference whenever practicable.

IV. DISEASES OF THE TONSILS.

Follicular Tonsillitis.

Synonym.—Lacunar Tonsillitis.

Definition.—A form of acute tonsillitis in which the inflammation involves the mucous membrane covering the glands and lining the crypts or follicles of the tonsils.

Etiology.—Children and young adults are more liable to be affected by this disease than elderly persons, and it is rare during infantile life. Exposure to wet and cold is the common cause, though bad hygienic surroundings, such as defective drainage, malaria, sewer-gas, etc., are believed to exert some influence in its causation. Some believe that there is a relationship between this disease and rheumatism—that the two are liable to coexist—and some even claim that follicular tonsillitis is a phase of rheumatism which affects children. While these are facts which lend support to the theory that rheumatism is allied, etiologically, to catarrhal disturbance, it is not probable that tonsillitis sustains any more relation to it than other acute catarrhal affections.

Pathology.—The mucous membrane lining the crypts is most severely inflamed, though that covering the external portion of the tonsil is also affected. The follicles exude a whitish material, consisting of pus-cells, epithelial cells, bacteria and mucus, which appears

at the mouths of the lacunæ as circumscribed white spots resembling the exudation of diphtheria in general appearance but differing from it materially in true character, as it lacks the fibrin found in the exudation of that disease.

Symptoms.—The constitutional symptoms of follicular tonsillitis are often out of proportion to the local trouble. There is usually an initiatory chill with rapid pulse, the temperature rising as high as 103° or 104° F. and sometimes higher. Muscular pain is a usual symptom in greater or less degree and is often marked. There is severe aching in the muscles of the neck and head, which may extend along the back, the muscular pain assuming the proportions of muscular rheumatism and demanding special therapeutic attention. The throat is now stiffened and swollen, the tonsils enlarged and their surfaces dotted with spots of creamy-white exudate occupying the mouths of the crypts, and they may be so abundant as to coalesce in some places, though this condition is not common. The cervical lymphatics are slightly though not markedly enlarged. Respiration is more or less impeded, the breath is foul and swallowing difficult. The tongue is loaded with a pasty-white fur, the bowels are constipated and the urine is scanty and throws down urates. Prostration is a marked symptom at first, though with proper treatment the weakness abates within three or four days, the swelling subsides and the fever and muscular pain disappear. Recurring attacks, however, are not uncommon upon slight exposure.

Diagnosis.—The diagnosis between follicular tonsillitis and mild cases of diphtheria is not always an easy matter. Diphtheria may begin in the follicles of the tonsil and the first appearance of the part may be that of lacunar tonsillitis. It is to be remembered, however, that the exudate of diphtheria spreads with greater or less rapidity and soon creeps along the tonsillar surface to the pharynx and spreads upon its walls. It is ashen-gray in color, in contrast with the creamy-white color of the exudation of tonsillitis. The enlargement of the cervical glands is also much more marked in diphtheria, while there is hardly ever so much elevation of temperature as there is in tonsillitis. Bacteriologists presume to differentiate with the microscope, and assert that the presence of the Klebs-Löffler bacillus is diagnostic of diphtheria, but my own experience has shown me that grave errors arise when this is made the principal means of diagnosis.

Prognosis.—Follicular tonsillitis is usually readily controlled by Eclectic treatment. Even a few hours of proper medication find the patient much more comfortable and a couple of days suffices to control nearly all unpleasantness, only slight local discomfort remaining.

Treatment.—*Aconite* and *phytolacca* will soon relieve the urgent symptoms. The following prescription, for an adult, is the proper

thing: ℞ Green-root tincture (or specific) phytolacca ʒi, Lloyd's aconite gtt. v–x, aqua ad ℥iv. S. Take a teaspoonful every hour. This will soon control the fever and assuage the local irritation, and the principal requirement then is to relieve the muscular pain. This is readily accomplished with *macrotys* or *rhamnus californica.* I prefer to use these agents in the form of decoction, in half or full wineglass doses, macrotys being preferable where the bowels are sensitive to the cathartic action of the rhamnus. One of these agents should be administered steadily until the muscular pain has subsided. Where the rhamnus causes catharsis or the cimicifuga causes headache the dose should be considerably lessened.

Periodicity is a common attendant of follicular tonsillitis in malarious districts, and satisfactory treatment demands that this element shall be properly managed. The antiperiodic action of *quinia sulphas* may here be sought by the common method, or three-grain doses of arseniate of quinia 3x, repeated every four hours for two or three days, may be used instead. When the patient is robust and the system is in a proper condition for the administration of quinia, it will produce the most prompt results. The arseniate is more pleasant, but requires a longer time to produce the effect.

The compound tincture of *guaiacum* or powdered gum guaiac is an excellent remedy in this disease, though with aconite and phytolacca we will hardly care to make use of it, as the combination is unequalled in pleasantness and efficiency for its effect on tonsillar inflammation of this character. Where the patient seems predisposed to frequent recurrences of the disease the protracted use of *baryta carb.* 3x will assist in fortifying the parts against later invasion.

Local applications are not of much use except for their mental influence, though sometimes this is not to be neglected. Thus, vinegar packs may be employed or even tepid-water packs. Hot applications might favor suppurative action, and are to be avoided.

Peritonsillar Abscess.

Synonyms—Quinsy; Amygdalitis.

Definition.—An inflammation of the connective tissue external to the tonsil.

Etiology.—Any of the causes of follicular tonsillitis may give rise to peritonsillar abscess, though it may arise from infection originated by that disease. When quinsy has once occurred, the subject is especially prone to later attacks upon slight provocation, a permanent susceptibility seeming to remain for years.

Pathology.—Suppurative inflammation occurs in the connective tissue surrounding the tonsil, the upper portion being usually affect-

ed, as the dense structure at the lower anterior part of the gland is more resisting. The abscess usually extends upwards between the pillars of the fauces and sometimes backward and downward along the posterior pillar.

Symptoms.—These are usually severe, the affected part being swollen and sensitive, that side of the neck being stiffened and enlarged externally. The jaws soon become so swollen as to prevent opening of the mouth, and deglutition is extremely difficult and painful. The voice becomes muffled and nasal, and complete inability to swallow often results from the extensive tumefaction about the fauces. Throbbing in the affected part begins early, the tensive pain being varied by alternate dartings in the middle ear. Chilliness at intervals heralds the advent of suppuration, which begins within two or three days after the initiation of the active symptoms; and the patient is now only able to open the jaws sufficiently to protrude the tongue with great difficulty. If examined early the tonsil will present a lateral tumefaction, which crowds the soft palate upward and and the tongue downward on the affected side and bulges into the opening of the fauces, sometimes nearly closing it, the mucous membrane presenting an angry, reddened appearance. As suppuration proceeds a prominent bulging point may be distinguished just beneath the soft palate of the affected side, indicating the near approach of pus to the surface.

The tongue is usually heavily coated with a pasty-white, offensive fur, the breath is fœtid, and constant accumulation of tenacious mucus in the throat gives rise to frequent hawking and other efforts to clear the passage. There is usually elevation of temperature, the thermometer often indicating from 102° to 104° F. The bowels are constipated, the urine scanty and high colored, the skin dry and husky and the patient is restless and uneasy, his sleep being noisy and stertorous.

Diagnosis.—Retro-pharyngeal abscess might be mistaken for quinsy, though palpation of the affected side at the angle of the jaw and examination with the finger within the throat will remove all doubt.

Prognosis.—There is some danger of escape of pus into the larynx at the moment of discharge, especially if it occurs with the patient in the recumbent position. Œdematous laryngitis is among the possible complications, and this may render asphyxia imminent. However, with good treatment a favorable termination may usually be anticipated.

Treatment.—An excellent prophylactic, where the quinsy-habit has become established, is the protracted use of *baryta carb.* 3x, and this is especially commendable upon the first appearance of the

symptoms of an attack. The early use of *potassium chloride* 3x is also commendable, as, if begun early, it promises to arrest the formation of pus and abort the disease. When follicular tonsillitis occurs coincidently, as is sometimes the case, *phytolacca* and *aconite* may be alternated with one of these remedies. Two grains of the 3x trituration of baryta carb. may be administered every two hours when used to abort the disease, or every three or four when used to fortify the tonsil against future attacks. If potassium chloride is to be used dissolve five grains of the 3x trituration in half a common tumbler of water and give a teaspoonful every one or two hours.

Early *puncture* with an aseptic bistoury is the best treatment after suppuration has been established. Even if pus be not already formed early evacuation of blood relieves painful tumefaction and the incision facilitates prompt escape of the earliest formation of purulent material. The puncture should be made near the upper portion of the tonsil, just below the soft palate, in a horizontal direction, nearly backward, rather toward the median line of the throat. In the absence of a tonsil bistoury a common straight bistoury may be guarded, except at the point, with a wrapping of linen or cotton cloth, and serve an equally good purpose.

Peritonsillar Abscess.
p, point for puncture.

The patient may be nourished, in event of inability to swallow, by injecting milk or other liquid food into the œsophagus with a Davidson syringe through a gum-elastic catheter.

Chronic Tonsillitis.

Synonym.—Hypertrophy of the Tonsils.

Definition.—Chronic enlargement of the tonsils, usually occurring in children.

Etiology.—Chronic tonsillitis may arise from repeated attacks of follicular tonsillitis or may come on insidiously. Members of certain families are especially prone to its development, those of lymphatic temperament being probably most liable. Children of syphilitic, tubercular or rheumatic history are often subjects of chronic tonsillitis. It is also liable to follow attacks of scarlatina, diphtheria, rubella, measles and other severe infectious diseases, especially those which are attended by faucial irritation. It is most common between the ages of three and five, though older children may be affected by it. After the age of fifteen there is a general tendency for the glands to undergo atrophy, the process being slow, however, and often continuing to the thirtieth year.

Pathology.—All the tissues of the tonsils increase in size, the number of lymphoid cells being especially augmented. The follicles become deepened and dilated, their orifices being visible to the naked eye upon superficial inspection, the gaping openings often disclosing the presence of a yellowish-white, offensive, curdy material within. The lax structure permits of the rapid growth of adventitious tissue in the tonsils, and rapid enlargement usually results when the hypertrophic processes begin, the fauces being soon blocked by the protruding organs.

Symptoms.—A subject of hypertrophic tonsillitis is constantly annoyed with a sensation as of a foreign body in the throat. The voice is muffled and husky and deglutition is impaired—though not severely, except when an attack of acute tonsillitis occurs in connection with it, which, however, is often the case. Respiration is through the mouth, and sleep is usually characterized by chokings and startings—which are many times alarming to parents, strangulation being frequently suggested at night, though not at all imminent. Hearing is often impaired from tumefaction about the orifices of the Eustachian tubes. Upon inspection the glands will be found dusky and swollen, obstructing the fauces to greater or less extent and presenting the gaping orifices with curdy contents as described under pathology, the breath being fœtid and offensive.

The habit of mouth-breathing entails more or less pharyngeal and laryngeal irritation, manifested by hawking, empty swallowing and hacking cough. Obstructed respiration and resultant defective oxygenation of the blood are liable to terminate in deterioration of the general health. Long-continued post-nasal obstruction may give rise to a stupid, dejected cast of countenance so peculiar to this form of tonsillitis, and the conformation of the thorax may finally become altered, the chest being flattened at the sides and bulged forward at the sternum.

Diagnosis.—Malignant disease of the part might be mistaken during its early stage for chronic tonsillitis, though the bright red color, severe pain and lateral character of malignant disease (one side being affected rather than both) would afford clearly defined diagnostic differentiation. As the disease advanced to a later stage there should be no chance for confusion.

Prognosis.—As the hypertrophy continues there is increased danger of permanent damage to the voice as well as to the general health. Even though atrophy may be expected to begin at puberty, it is not advisable to neglect the present condition. There is increased liability to such infectious diseases as diphtheria, scarlatina, etc., when the follicles are enlarged, and treatment for a radical cure is therefore additionally important.

Treatment.—Certain remedies influence the tonsils and reduce enlargement in hypertrophied conditions. *Baryta carb.* is excellent for this purpose, two or three grains of the 3x trituration three or four times daily answering a good purpose when persevered in for several months at a time. The *iodide of barium* is also an excellent remedy employed in the same manner. A reliable auxiliary is the *galvanic current*, which may be applied twice weekly by holding a positive electrode against the tonsil in the throat and the negative sponge upon the outer surface over the affected region. The patient will be able to bear the current only a second or two at a time, but it may be repeated two or three times at each sitting. Interstitial injections of *ergot*, *thuja* and *iodine* into the tonsils have been successfully used by some. Perhaps the local application of *galvano-cautery* may prove more reliable still, deep cauterization being followed by cicatrization and contraction of the part. When such measures fail, *amputation* of the organs may be in order, the operation being simple and of little danger, while loss of the tonsils involves no serious results to the general system. A small volsellum forceps may be used to steady the diseased part while a strong scissors (curved on the flat) is employed to excise it. Or, a tonsil bistoury may be used instead. The Matthieu tonsillotome is an excellent instrument for removal of the tonsil, when intelligently employed. The operation is so simple that the ordinary practitioner need not hesitate to attempt it, little hemorrhage following, though in small children anæsthesia may be necessary.

Adenoid growths upon the vault of the pharynx (the third tonsil) are sometimes complicated with chronic tonsillitis and require removal as well as the tonsils. A special cutting forceps is manufactured for this purpose.

Dr. H. W. Kendall, of Quincy, Ill., describes a method which he has used with advantage for ten years: "We have an efficient cauterant and at the same time an antiseptic and alterant in pure hydrochloric acid, which is always friendly to human flesh. This is the agent that I have found so efficient in reducing enlarged glands in all parts of the body, but the method of using it is the particular point that I wish to present in this short paper. My method is the use of capillary glass tubes (Bohemian or Whitall & Tatum's glass) one-eighth of an inch calibre heated in a Bunsen flame and drawn to a point, the shaft of the drawn part two inches long, with calibre one-sixty-fourth of an inch, broken off and fire polished. Now, if the shaft of the tube is five inches long the drawn part will hold, after dipping in the fluid, one minim; if the larger shaft is increased in length it will hold more. When the point of this tube touches any

substance it will deposit a fraction of the drop; by long contact it will deposit all that it contains.

"I dip these tubes into pure fuming hydrochloric acid and push them into the excretory ducts of the glands, three in each gland at each sitting twice a week. This operation is painless and produces no inflammation or swelling. Five or six applications are sufficient for moderately enlarged glands."

V. DISEASES OF THE ŒSOPHAGUS.

Œsophagitis.

Synonym.—Inflammatory Dysphagia.

Definition.—A catarrhal inflammation of a part or the whole of the mucous membrane of the œsophagus.

Etiology.—Acute inflammation of the œsophagus is usually due to the action of acrid fluids or solids in their passage to the stomach. The incautious swallowing of scalding fluids, such as hot chocolate, coffee, or of hot food, occasionally causes it. Children sometimes swallow lye or carbolic acid, or the latter substance is taken with suicidal intent. The accidental lodgment of spiculæ of bone, artificial teeth or other foreign bodies in the œsophagus, sometimes results in inflammation of acute character, which is followed by long-continued subacute or chronic inflammation. The excessive use of alcohol, extension of pharyngitis or other inflammation of the throat to the œsophagus, tuberculosis and other exciting and predisposing causes might be named. Chronic œsophagitis may follow an acute attack, be developed from a tuberculous or syphilitic condition or be due to the irritation of a foreign body lodged along the tube. Diphtheritic infection, scarlatina, cholera, pyæmia, septicæmia or other infectious diseases may give rise to membranous œsophagitis.

Pathology.—In acute œsophagitis the mucous membrane is highly reddened and covered with a layer of muco-pus and detached epithelium, and the tissues are swollen and softened. In the chronic form the mucous membrane is darkened to a slaty-blue color, the submucous tissue is swollen and the surface is covered with a thick, tenacious mixture of mucus and pus. The œsophageal walls generally are thickened, and a part or the whole of the tube above the location of an ulcer, which will mark a narrowing, will be dilated or there may be several constrictions and dilations. Sometimes extensive diverticula are formed and there may be hernia of the mucous membrane through the muscular wall, with final perforation. The ulceration usually occurs at the seat of most prominent irritation.

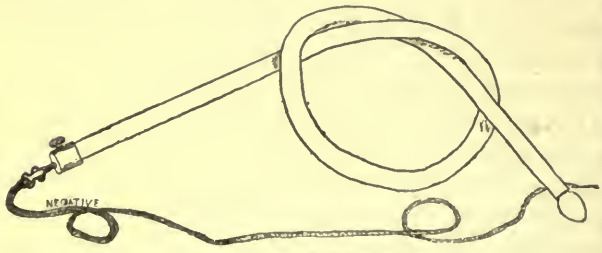
In membranous inflammation the morbid changes common to such inflammation upon other mucous surfaces will appear.

Symptoms.—In acute œsophagitis burning and gnawing sensations are experienced along the œsophagus, behind the sternum, through the mediastinum and between the shoulders, aggravated by attempts to swallow even the most bland liquids. Extreme thirst, great depression and anxiety, attended by slight febrile disturbance, are present in the acute form. In chronic œsophagitis there is not much pain except when solids are swallowed, though if ulceration exist there is occasionally vomiting of vicid mucus mixed with pus and tinged with blood.

Diagnosis.—Knowledge of the provoking cause will enable the practitioner in acute cases to arrive at a correct diagnosis. Chronic œsophagitis may be confounded with cancer, but here the severity of the pain, which is severe when the part is at rest, the rapid progress of the symptoms and the marked cachexia, will render the diagnosis clear.

Prognosis.—In acute œsophagitis the damage to the stomach and other associate organs should be taken into consideration in summing up the probable outcome. In croupous œsophagitis the systemic condition of the patient—the severity of the attending case—will determine, more than the local condition, the probable result. Chronic and subacute œsophagitis, where deep-seated stricture is not present, are amenable to curative treatment.

Treatment.—Where œsophagitis is due to the presence of foreign bodies in the œsophagus their removal is the first matter to be considered. Inflammation of acute character should be treated with *veratrum* or *jaborandi* in ordinary sedative doses. *Echinacea* may be advantageously combined with either or both these agents. *Bichromate of potassium*, in the 2x or 3x trituration, acts well in subacute inflammation, and *lachesis* or *naja*, in homeopathic attenuations, may relieve the burning, stabbing pain. After the acute symptoms have subsided a flexible galvanic electrode, consisting of an elongated shaft of spirally coiled brass spring-wire (eighteen or twenty inches in length) covered and insulated with a section of rubber tubing, having a bulb-shaped nickel- or silver-plated metal terminal at one end and a screw clamp attachment at the other, connected with the negative cord, may be passed down-



Œsophageal Electrode.

ward and upward along the œsophagus twice a week (two or three times) with a current of from five to ten milliamperes (or eight to sixteen cells of an ordinary portable carbon-zinc battery). This is an excellent means of permanently relieving the irritation and excoriation. The patient should take the positive pole, consisting of a wetted sponge, in the hand only after the bulb has passed into the œsophagus, as unpleasant shock is thus averted.

A liquid *diet* should be used, and movement of the part avoided so far as possible.

Obstruction of the Œsophagus.

Etiology.—This condition may arise from a variety of causes, which may be arranged under the following heads: (1) Those which are due to organic changes in the walls of the œsophagus from cancerous infiltration; hypertrophy of the coats generally from inflammatory action, the submucous coat being generally involved; fibroid changes due to chronic inflammation; localized thickening due to cicatrization after wounds, lesions and ulcers; syphilitic disease. (2) External pressure from various causes, such as bronchocele; enlargement of the cervical or thoracic lymphatics; cancerous or fibroid tumors; aneurisms; abscesses; great tension of the pericardium with fluids. (3) Growths within the œsophagus, such as fibroid tumors, etc.

Pathology.—Obstruction at any point finally gives rise to dilatation and hypertrophy of the œsophagus above, accumulation of food tending to distend the walls of the part, while the necessary resistance favors thickening of the muscular structure. Continued distention, however, may result in rupture of the muscular wall with hernia of the mucous membrane; or ulceration may finally occur at some point, with possible perforation.

Symptoms.—Difficulty of swallowing is the most prominent symptom, pain and sensation of stoppage occurring when the food reaches the point of obstruction, this most commonly being behind the upper portion of the sternum. The disease is progressive, the difficulty of swallowing becoming more and more marked until it is impossible for even the smallest particle of solid food to reach the stomach. Liquids and soft, pulpy food-substances pass the obstruction best. When food cannot pass it is soon rejected, either by gradual regurgitation or sudden spasmodic action; or, being retained for a time, it is discharged in large quantities of alkaline, sodden material, mingled with mucus and pus and, perhaps, tinged with blood. If the condition be due to cancerous infiltration severe pain usually attends, both during the taking of food and between

times as well, and debility, emaciation, waxy color and other symptoms of cancerous cachexia develop. In ordinary ulceration there is not much pain except during efforts at deglutition, and not even then unless the ulcer be irritable. Another cause of emaciation and debility besides that of cancer may be that of starvation from inability to swallow sufficient food to meet the demands of the body. If perforation occur sudden collapse or symptoms of septicæmia may follow.

Treatment.—Soft strictures—those involving only the mucous and submucous structures—may be benefited if not wholly relieved by the patient use of graduated dilators, employed at intervals and passed cautiously to avoid irritation. In the beginning anæsthesia may be employed and afterward a full dose of *bromide of potassium* may be administered an hour beforehand to quiet irritability and facilitate the operation. Cicatricial strictures will not yield to such treatment, however, and had better not be irritated by efforts to dilate them. Attention to the diet and such other palliative measures as individual cases may demand are all that can be adopted, unless a gastric fistula for the introduction of food directly into the stomach is established.

Cancerous stricture should not be disturbed, as irritation usually augments the rapidity of its development; all that we can expect to do here is to relieve the pain which attends, and we may accomplish this usually with ten- or fifteen-drop doses of *echinacea*. *Silica* 3x is an excellent remedy for this purpose, three-grain doses of the trituration being used three or four times a day, though it is second to echinacea in value. Where the ulceration which causes the obstruction is non-malignant a stomach-pump may sometimes be used successfully in introducing nourishment.

Chelidonium majus has recently attracted attention as an internal remedy for curative effects in cancer generally. Cures of unmistakable cancer of the œsophagus and stomach have been reported by apparently respectable authority. Small doses, frequently repeated, for a long time, were required to remove the morbid growths.

Syphilis, bronchocele, hydropericardium and other special causes of œsophageal obstruction will demand special treatment, adapted to the condition present.

Functional Disease of the Œsophagus.

Paralysis of the œsophagus sometimes attends diphtheria and hysteria, and arises from glosso-pharyngeal paralysis and general paralysis of the insane. It also attends progressive muscular paralysis and certain diseases of the brain.

Symptoms.—Dysphagia is the only prominent symptom. The food may escape and pass into the larynx, producing serious respiratory embarrassment, this being especially true of liquids. Deglutition is facilitated by the erect posture.

ŒSOPHAGISMUS, or spasm of the œsophagus, may occur in hysteria, in hypochondria, in muscular rheumatism or from an irritable ulcer in the passage. It may also be caused by the bolting of large lumps of solid food, by swallowing extremely hot or cold food, or by the abuse of alcohol. Irritation of the œsophageal nerves may also be a cause, and dyspeptic symptoms are sometimes attended by it. Rectal irritation, lacerated cervix uteri, adherent prepuce or clitoris and other orificial lesions may be accountable for it.

Prominent among the *symptoms* is a sensation of obstruction as from a solid substance in the gullet; and when food is taken there is stoppage of the bolus upon swallowing at some point in the passage. These symptoms are temporary and appear erratically, as the exciting cause provokes spasmodic action. There may be quite long intervals during which there is no difficulty whatever in swallowing. If a bougie be passed while the spasmodic action is on its course is arrested at the point of contraction, but if steady pressure be maintained against it at the place of resistance there is soon yielding and the instrument passes the obstruction easily. During severe attacks there is a sensation of constriction and suffocation about the throat due, probably, to spasm of the cervical muscles. When it occurs in muscular rheumatism it is due to metastasis of the rheumatic affection, pain then attending the sense of obstruction and suffocation. The upper portion of the œsophagus is usually involved. Little constitutional disturbance is present, the patient appearing well nourished, though dyspeptic, hysterical and hypochondriacal symptoms are common in most such cases.

Treatment.—*Bromide of potassium* allays spasm and hyperæsthesia of the œsophagus and is almost always an appropriate remedy in œsophagismus. From ten to twenty grains may be administered every two or three hours while the active symptoms continue. It rarely affords permanent relief, but it is useful to relieve speedily. Markedly hysterical cases should receive specific *gossypium* in tendrop doses four or five times daily until the spasmodic symptoms subside. *Valerianate of ammonium*, tincture of *valerian* and *valerianate of zinc* are all useful. *Naja* relieves spasm of the œsophagus where the œsophageal nerves are irritated. Rheumatic complications demand *cimicifuga* or *rhamnus californica*. *Faradism* often does good, the positive pole being applied to the tongue with a metal tongue-depressor while a wetted sponge attached to the negative is held against the epigastrium.

In all these cases careful inspection of the rectum, cervix uteri and other orifices should be made to detect any irritation there, and if this be found present the removal of the difficulty by proper orificial surgery is the correct course to pursue.

VI. DISEASES OF THE STOMACH.

Acute Gastritis.

Synonyms.—Acute Gastric Catarrh; Acute Dyspepsia.

Definition.—An acute inflammation of the mucous membrane of the stomach.

Etiology.—Ordinary causes of inflammation of the mucous membranes generally, such as sudden arrest of secretion from cold while the person is relaxed, may excite this condition in people who are delicate in respect to the state of the digestive organs. The action of hot or cold articles of food is calculated to bring on acute gastritis, and certain chemicals, such as alcohol, strong acids, arsenic, etc., may excite a high grade of inflammatory action when allowed to enter the stomach in concentrated form. Some diseases incline to bring about the condition; as, for instance, cholera morbus, cholera, yellow fever, etc. Acute gastritis attended by febrile symptoms sometimes occurs epidemically. The predisposing conditions are disordered states of the gastric mucosa, which place its vitality below par, as in elderly persons or delicate women and children, in whom the stomach is in an enfeebled condition, the disease being here provoked by indiscretions in diet.

Pathology.—There are different grades of acute gastritis. Exposure of the mucous membrane of the stomach through a gastric fistula has afforded opportunity to watch the condition of the mucous membrane during life when inflamed, and it has been seen that the part is reddened and that a coating of mucus is thrown out over the reddened surface. The redness in moderate cases occurs in patches, the hyperæmia being punctiform or capillary in character; but when irritant poisoning occurs the entire mucous membrane is highly reddened and swollen, that upon the summits of the rugæ suffering most. Small extravasations may appear, and minute ulcers and follicular erosions are not uncommon. In very severe cases suppuration and sloughing of the submucous structure may occur. The peptic and mucous glands are also involved, the cells and nuclei being enlarged and increased in number, the tubules being elongated and prominent. The secretion of gastric juice is thus interfered with, an alkaline, ropy mucus covering the surface. The lymphoid elements between the glands are increased in number and hypertrophied.

Many mild cases of gastritis occur in which such marked changes are absent, a punctiform redness of the mucous membrane with increase of catarrhal secretion being the extent of the morbid condition.

Symptoms.—These vary widely, according to the severity of the inflammation. In mild cases there are symptoms of indigestion, such as unpleasant sensations in the epigastric region—burning, nausea, eructations, and vomiting which is followed by relief. Constipation may attend, though there is sometimes diarrhœa, especially in children. The tongue is coated and there is an excessive amount of saliva, attended by metallic taste. Such cases subside within twenty-four hours usually, as they result from slight causes and depend on individual susceptibility. Severe cases are marked by burning pains, often of excruciating character, which invade the epigastric region and radiate throughout the entire abdomen, colicky sensations alternating. The inflammation may be marked by a chill and febrile action (102°—104° F.). Vomiting is a common symptom and is repeated frequently, the tongue being furred and the breath offensive. Food is ejected at first, but bile mixed with mucus and watery fluids appears in the ejections later, there being absence of hydrochloric acid, a presence of lactic and fatty acids and a superabundance of mucus. The urine presents the usual characteristics of febrile action, an abundant deposit of urates being thrown down.

In gastritis from toxic poisoning there is burning pain in the mouth, throat, œsophagus, stomach and bowels, watery diarrhœa (when arsenical), ptyalism, difficult deglutition and frequent vomiting (the ejections containing blood and sometimes portions of mucous membrane), while the abdomen is swollen and tender upon pressure. In extreme cases collapse may occur, the pulse being thready, the respiration labored, the skin cold and covered with clammy sweat and the patient extremely restless and anxious.

Diagnosis.—When the disease occurs without any well-pronounced cause, it may at first be mistaken for some form of infectious fever announced by active gastric irritation, as is sometimes the case in scarlatina and other infections. Later developments, however, will distinguish it from these affections. When irritating drugs have been swallowed a knowledge of this fact will enable us to decide as to the character of the complaint. The experience of delicately constituted persons who suffer from gastritis from slight causes will assist the physician in most cases in arriving at a correct diagnosis.

Prognosis.—Such cases as are marked by dietary indiscretions usually recover within two or three days. Others may run a still more violent course, depending on the gravity of the exciting cause, a chronic gastric catarrh being finally established. Poisoning with arsenic or other corrosive poisons is liable to run a still more severe

course, the symptoms of toxic poisoning being sometimes of grave and fatal character.

Treatment.—In mild cases of acute gastritis diluents may be used to favor emesis. These may be warm water, flax-seed tea, slippery-elm water or some other soothing agent. Following this a decoction of the bark of peach-tree shoots, a weak infusion of *hydrastis* or (what is better) the following prescription may be administered: ℞ Green plant tincture rhus tox. gtt. xv, specific ipecac gtt. x, water ʒiv. M. Sig. Take a teaspoonful every hour. Sometimes two grains of *subnitrate of bismuth* every hour will serve a better purpose. For the treatment of poisoning the reader is referred to works on toxicology.

Phlegmonous Gastritis.

Symptom.—Suppurative Lenitis.

Definition.—A suppurative inflammation of the submucous (areolar) tissue of the stomach.

Etiology.—This disease is rare and usually occurs between the ages of twenty and forty years. It may arise from the infection of pyæmia, septicæmia, typhoid or typhus fever, or diarrhœa as a secondary affection or may occur idiopathically. Traumatism may be an occasional cause.

Pathology.—The inflammation may be circumscribed or diffused. The wall of the stomach at the point of inflammatory action is thickened, œdematous and friable, with infiltration of the areolar tissue with sero-fibrinous and purulent material. In the diffused form the mucous membrane may be thinned and perforated in numerous places, affording exit to pus from a variety of irregularly shaped cavities located in the submucous tissue. The mucous surface is reddened, sometimes dusky in hue, and gangrenous spots may appear in various places. Sometimes the peritoneal coat is involved, the condition then assuming the characteristics of acute peritonitis. Pus may perforate this membrane and escape into the peritoneal cavity. Gastric ulcers may arise from perforation of the mucous membrane.

Symptoms.—Active febrile symptoms usher in this disease. There is a chill, followed by febrile reaction, the temperature rising as high as 104° or 106° F. There is intense pain in the epigastric region, with loss of appetite and consuming thirst. Persistent vomiting of a dark colored, bitter fluid containing more or less pus succeeds early upon the initial chill, the patient is rapidly prostrated and becomes anxious and watchful. Delirium with jaundice soon follows, and typhoid symptoms with muttering, wandering or stupor precede the

period of collapse (which is soon developed), the patient dying in a comatose condition.

Diagnosis.—This is very difficult, and the disease is usually unrecognized during life, autopsies supplying the most that has been known upon the subject.

Prognosis.—This is always unfavorable. The majority of cases prove fatal within the first week, especially if the inflammation be diffused. Circumscribed inflammation here may not prove so rapidly fatal, the patient surviving for two or three weeks. Secondary abscess of the liver and peritonitis may attend.

Treatment.—This is principally palliative, hypodermic injections of *morphia* being most reliable to relieve the pain. Hypodermic injections of *echafolta* might be tried for the control of the inflammatory action and to prevent extreme destruction of tissue.

Parasitic Gastritis.

Fungi occasionally develop in the stomach and excite inflammation. Sarcinæ and yeast fungi are probably perpetuating causes of chronic gastritis, and occasionally a case of acute gastritis seems ascribable to the presence of parasitic growths. The anthrax bacillus has been known to develop in the gastric mucous membrane and Klebs has described a bacillus gastricus which develops in the gastric tubules and excites acute gastritis. The larvæ of certain insects has been known to cause gastritis. Diphtheria, tuberculosis and syphilis may attack the gastric mucous membrane.

Chronic Gastritis.

Synynoms.—Chronic Gastric Catarrh; Chronic Dyspepsia.

Definition.—It is a disturbance of digestion attended by an excess of gastric mucous secretion, with vitiation of the digestive juices from fermentative products, and finally alteration of the structural integrity of the stomachal walls.

Etiology—The causes of this condition are numerous and varied in character, but all tend to one result—difficult and protracted chymification, with formation, within the stomach, of fermentative material. This may result from acute gastritis, though it is more apt to come on from indiscretions in diet, such as the use of highly seasoned or indigestible food; irregular and hasty eating; gourmandizing; addiction to the excessive use of tea, coffee, alcohol or tobacco or from the habit of using iced foods or drinks during meals. It may develop from rectal, prostatic or uterine irritation and such constitutional diseases as gout, anæmia, chlorosis, tuberculosis and dia-

betes, and malarial cachexia may be attended or preceded by it. Pulmonary tuberculosis or chronic interstitial nephritis may be heralded for months by gastric catarrh long before the pending causal disease has been fully developed, slow and inadequate digestion encouraging the growth of the yeast plant, sarcina and other elements of gastric fermentation, thus giving rise to and perpetuating irritation of the gastric mucosa. Portal obstruction, by causing engorgement of the gastric capillaries, may retard digestion and, finally, through disturbance of the functions of the gastric tubules and resultant slow and feeble digestion, permit the accumulation of sufficient provoking cause to bring on a chronic catarrh, it therefore being frequently associated with active or passive hepatic congestion, hepatic cirrhosis, splenic hypertrophy, pancreatic disease or cardiac or pulmonary engorgement. Also, it may be associated with various local diseases of the stomach, such, for example, as cancer, ulceration or dilatation.

Pathology.—Pathologists recognize two forms of chronic gastritis: (1) The simple or common, and (2) the sclerotic—the second being rare. The *simple* form is attended by hypertrophy of the intertubular mucous membrane with consequent choking out of the gastric tubules; while the second is attended by atrophy of the entire mucous membrane as well as of the secreting structures. The first is marked by profuse secretion of mucus with restriction of the normal amount of gastric juice, the lining of the stomach being covered with a tenacious coating which mechanically and chemically interferes with normal chymification, while the second is characterized by dryness and lack of secretion not only of gastric juice but of mucus, the organ being dilated, its walls thinned and atrophied, with fatty degeneration of its glandular elements. In another sclerotic form (which is exceedingly rare) there is fibrous degeneration of the gastric walls, the muscular structure being thickened with fibrous growth from hyperplasia of the connective tissue until the coats are contracted and hardened, the viscus being lessened in size and concentrated until its outlines may be traced by palpation through the abdominal walls.

In the common form removal of the tenacious gray mucus covering the interior of the stomach will reveal more or less alteration of structure in the mucous membrane, the amount and character depending upon the duration and severity of the disease. Œdematous spots covered with granulations, ecchymoses and more or less extensive areas of pigmentation are distributed over the surface. Thickening of the mucous membrane is prominent, especially about the pylorus, and this may be so extensive as to obstruct the opening, the stenosis resulting in gradual dilatation—gastrectasia. In some cases the sub-

mucous tissue is implicated, the thickening being attended by infiltration of the structure with migrating connective-tissue cells and development of adventitious fibrous growth, which renders the walls firm and unwieldy and interferes greatly with normal peristaltic action. Mammillation of the surface of the mucous membrane due to obstruction of the tubules by pressure from intertubular hypertrophy, and consequent distention by accumulation of their secretions until they stand out prominently, may sometimes be observed. Another form of mammillation is that which attends hypertrophy of the peptic glands, this resulting in an increased area of mucous surface, which being more voluminous than the basement membrane is thrown into folds or corrugations. As the disease continues the muscular coats may become still more involved in the thickening process and the peristaltic movements will become further impeded. Finally, the serous layer may be involved and adhesions occur between opposing surfaces of the reflected peritonæum.

In long-standing cases the mucous membrane near the pylorus is very liable to be the seat of abrasions, superficial ulcers of circular shape, varying from half an inch to an inch in diameter, occupying this region, the intervening mucous membrane being reddened and œdematous. The ulcers are superficial, rarely extending deeper than the mucous membrane, their bases being covered with mucous cells, epithelium and nuclei. Minute points of ulceration may appear, scattered about over the entire surface, marking the locations of similarly affected solitary and lenticular glands. The inflammation usually extends to the duodenal mucous membrane, similar changes occurring here, and the common bile duct may be involved during aggravations, icteric symptoms from obstruction at various intervals signalizing such complication. Dilatation of the stomach may attend some cases and contraction of its walls others, as varying pathological changes predominate. Amyloid degeneration of the walls of the stomach, secondary to waxy changes in the liver and spleen, may occur in advanced stages. Fatty infiltration of the tubules is detected under the microscope, and occasionally fatty degeneration of the tubular structures.

Symptoms.—Indigestion is the prominent symptom, a great variety of unpleasant accompaniments being liable to attend. Heartburn, associated with weight and fullness in the epigastric region, follows eating and continues for hours as soon as the disease develops. Later on there is actual pain of a burning, acrid nature, attended by eructations of gases and fluids, sour risings and tenderness on pressure over the epigastrium. Darting pains radiate from the stomach into the pectoral region and backward toward the scapulæ, and these may be aggravated by pressure over the epigastrium.

Burning along the œsophagus and in the throat and mouth, with increased secretion, is common, the lips and tongue sharing in the unpleasant sensation. The tongue is often red and slick, the papillæ being elevated and the tip pointed, though in other cases the general appearance of the organ may be normal. Craving for food (boulimia) is a frequent symptom, this amounting in many instances to an almost constant, unsatisfied, gnawing sensation in the epigastrium, though a small portion of food may satisfy it for a short time, during which the torments of difficult and painful digestion are experienced. A metallic taste is frequently present between periods of eating.

In aggravated cases vomiting is a frequent symptom, the material ejected consisting of partially digested food mixed with a large quantity of mucus, among which may be detected sarcinæ ventriculi, torulæ and varieties of bacilli and micrococci. There is absence of hydrochloric acid here in most cases, lactic acid, associated with butyric or acetic acid replacing it, though in rare cases there may be excessive secretion of hydrochloric acid. Digestion is necessarily retarded under these circumstances, and if the stomach be irrigated and siphoned seven hours after eating undigested food will still be found in the washings.

MICROSCOPICAL DEBRIS FROM CATARRHAL STOMACH.
a, sarcinæ ventriculi.
b, yeast plant.
c, bacteria and cocci.
d, epithelial cells.
e, leucocytes.
f, starch granules.
g, fat globules.
h, muscular fiber.
i, fat needles.
k, vegetable cells.

The bowels are usually constipated, though the reverse may be the case, undigested food then passing through the intestinal canal soon after it is swallowed (lientery).

Cardiac palpitation frequently attends the digestive process and the tumultuous throbbing may seem to be communicated to the sensitive stomach, accumulation of gases aggravating the difficulty and eructation affording only temporary relief. Stitching pains in the cardiac region may be added to the tumultuous action and vertigo is often associated with it. "Stomach cough," due to pharyngeal irritation partly and partly, in many cases, to voluntary efforts of the sufferer in seeking relief from præcordial oppression and epigastric discomfort, often attends.

Among the sympathetic symptoms are headache, langour, melancholy and emaciation. Where atropy of the gastric tubules is present anæmia is prominent.

Diagnosis.—The use of the stomach-tube will afford the best

means of diagnosis. If siphonage be practiced an hour or so after eating hydrochloric acid will usually be absent, and lactic acid associated with fatty acids appears, a large amount of mucus being present. If siphonage be practiced seven hours after eating, undigested food will be found still remaining in the stomach, while in cases of functional dyspepsia it will have disappeared. Malignant disease will be excluded by lack of cachexia, absence of perceptible tumor upon palpation and by the character of the vomit, coffee-ground material soon appearing in cancer. In gastric ulcer a diagnostic feature is hematemesis of bright blood.

Prognosis.—Chronic gastritis will usually improve readily under rational treatment, unless there be associated with it gastric ulcer, cancer, gastrectasia or organic, hepatic, renal or pulmonary disease. When neglected it may continue for years and eventually terminate in ulcer or pyloric stenosis, with resultant perforation or dilatation. A sympathethic disease of the supra-renal capsules is not an unfrequent complication, the supra-renal bodies seeming to sustain a peculiar relation of this nature to gastric irritation. The marked emaciation which attends long-continued cases renders the patient susceptible to attacks of acute disease and he is liable to succumb suddenly to some onset of this kind, to hematemesis or to the immediate results of pyloric stricture.

Treatment.—An important part of treatment is the abandonment, so far as possible, of all exciting or perpetuating causes. If the subject has been in the habit of using alcoholic liquors he should do away with them at once and forever. As a substitute three parts of Howe's *viburnum cordial* and one part of specific *avena sativa* should be combined, and recourse be had to this mixture in acceptable doses repeated as often as necessary until all depression and craving for the accustomed stimulant have passed away. A habit of using ice-water or iced drinks or foods should be dispensed with under all circumstances, warm foods and drinks of bland and digestible character being most applicable to restorative processes. Overeating and the use of objectionable food should be avoided—and this will be no easy trial for the patient if he be permitted to dine in the company of healthy persons, as the food they may be accustomed to may not be applicable to his case, and the power of association may be so strong as to lead him to transgress again and again to the complete defeat of curative measures. It will be better for him to eat alone and confine himself to small quantities of judiciously selected foods taken at shorter intervals than in health, with slow mastication. Fats and carbo-hydrates should be generally avoided as well as pastries, griddle-cakes and cheese.

Sometimes, when there is nephritic or cardiac complication (and

even in severe cases without complication), a milk diet adhered to strictly for several weeks will afford the best results. In order to prevent the formation of hard curds the milk should be diluted with soda-water, lime-water or other alkaline fluid. Where there is atrophy of the peptic glands pancreatized milk will be more appropriate. Sometimes, when the stomach is very weak and the milk causes nausea, it may be necessary to remove the cream before it is taken. Many persons will prefer butter-milk, and this may be allowed freely in such cases. From one to two quarts of milk or butter-milk may be taken every twenty-four hours, four ounces being allowed at a time with two-hour intervals, the amount being gradually increased and the intervals lengthened as improvement succeeds.

When milk does not sustain the strength (though such cases will be rare) underdone beef or, what is better, raw scraped beef may be allowed in connection with it, one or two ounces at a time two or three times a day being sufficient, though the amount may be increased as the patient's ability to digest food improves. Broths and soups should be avoided and tea, coffee and cocoa should be taken sparingly if at all and without milk or sugar. Oysters raw, broiled or panned are allowable and also stale bread without butter or with but a sparing quantity. Where there is an excessive amount of hydrochloric acid secreted the patient will live best on rare roast beef, rare steaks or the breast of chicken eaten with stale bread. Eggs should be thoroughly cooked for such persons and will then be well tolerated. If an egg be boiled for an hour the yolk, with a little salt added, will agree with the most delicate stomach. Where there is a strong tendency for food to undergo decomposition in the stomach salted and smoked meats and fish may sometimes agree better than other articles of diet. Here cream codfish, dried beef, jerked venison, caviar, etc., may be carefully tried in succession in small quantities, that the diet be varied. Cured meats may be employed for the manufacture of cream gravy to be eaten on toast or stale bread, the solid part being rejected.

Confusion as to a proper course of diet may be avoided if the patient can be induced to adhere to a *strictly dry diet.* This should consist *only* of stale bread, to be taken *ad libitum* with two or three ounces at a time of plain claret, which should not be repeated oftener than five or six times each twenty-four hours. No tea, coffee, water, milk or other fluid should be allowed, and no butter, meat or other food, except the plain, stale bread, should be consumed. This may seem a hardship at first, but adherence to the regimen brings abundant satisfaction by the end of ten or twelve weeks. There will be a provoking thirst for the first few days, after which this source of annoyance will have subsided. Upon this allowance

the patient will not over-eat and, though he may become emaciated and weakened somewhat, he will not starve, and will recuperate rapidly when a gradual return to ordinary diet is allowed, while the gastric disturbance will have subsided—if other proper measures have been applied in the meantime.

Saccharine, starchy and farinaceous foods are almost certain to undergo lactic and butyric acid fermentation in the stomach before their digestion can be completed, producing flatulency with eructation of gases and sour fluids. A person afflicted with chronic gastritis should endeavor to live carefully and abstemiously after recovery throughout his life, as it is not a difficult matter to provoke a return of the disease. During treatment, business cares and all other responsibilities should be avoided, that no expenditure of energy be made in an unnecessary direction, neither physical nor mental exertion being conducive to improvement. The case of Louis Cornaro, the Venetian, is not to be forgotten in this connection, for it illustrates the remarkable effect of careful living upon those seemingly hopelessly affected with gastric derangements.

Though my information does not justify me in asserting that he was a sufferer from chronic gastritis, the narrative suggests such a condition. Born with wealth, he was endowed with means and leisure to abandon himself to high and prodigal living; but a weak constitution, broken down at the age of thirty-five from riotous living and other excesses, rendered life a burden to him. The next five years were passed in almost constant misery, and at the age of forty his physician informed him that nothing could prolong his life more than two or three years, and temperate habits were advised as the means to relieve his sufferings during that time. He now began to gradually reduce the amount of food, both liquid and solid, consumed, until he at length took only what nature absolutely required. This, according to his own statement, was a difficult course to pursue and he often relapsed to over-eating; but he finally succeeded (within a year) in adopting permanently a spare and moderate system of diet, and was, at the end of this time, already restored to perfect health.

Being now an enthusiast, he proceeded from moderation to abstemiousness and diminished his daily allowance until the yolk of an egg sufficed him for a meal. Health and spirits improved and he soon became able to derive more pleasure from a small meal of dry bread than the most tempting viands of a richly-laden table had afforded him in his days of excesses. Such a course persevered in, with the avoidance of extremes of heat and cold, enabled him, after almost ending his life at thirty-five, to recuperate and become a centenarian. Modern experiences often acquaint us with similar cases, where individuals in desperation, after a prolonged treatment for

indigestion without benefit, recover under prolonged self-imposed starvation.

Constant and prolonged fermentation is the principal factor in the perpetuation of the disease, and the cleansing of the stomach of mucus and fermentative products is the direct way out of the difficulty. Modern times have afforded us superior advantages in this respect, and there is now little difficulty in curing uncomplicated cases of chronic gastritis even of long standing. If complications exist they should be removed if possible and the problem then becomes as clear as ever. The tenacious mucus, which serves as a nidus for fermentative products, must be removed and the interior of the stomach kept cleansed, when a little other treatment, except a proper regimen, is required. We possess two effective measures for this purpose, which may be employed singly or combined. I refer to (1) lavage and (2) disinfection and cleansing with hydrozone.

Lavage is an efficient means of cleansing the stomachal cavity. It is performed by the aid of an elongated, soft-rubber tube, to one end of which is attached a glass funnel. Dealers in rubber goods furnish these tubes upon application, with open lower end, fenestrated sides, and raised ridge to indicate the point of sufficient introduction, this being at the lips when the tube is *in situ*. In order to introduce it it is first coiled in a bowl containing warm or cold milk, according to the preference of the patient, and the fenestrated extremity is

LAVAGE: IRRIGATION AND SIPHONAGE.

then passed over the protruded tongue into the lower part of the pharynx, the patient assisting its onward motion by efforts at swallowing accompanied by deep inspirations. Steady pushing will now carry the instrument into the œsophagus and it will then glide easily along until the lower end passes into the stomach, when the funnel should be affixed. It may be necessary for the physician to assist

in the introduction for four or five times, after which the patient will be able to attend to it for himself. After initiation the patient holds the funnel in his left hand and a flask of the fluid to be used in the right, fills the funnel and raises it above his head, when the contents flow into the stomach (irrigation). The funnel is immediately afterward depressed below the level of the stomach, when the principle of siphonage operates to withdraw the liquid contents, which are allowed to flow into a pail placed between the patient's feet. Lavage is therefore divided into two stages, viz.: Irrigation and siphonage.

Reflex irritation, such as nausea and vomiting with dyspnœa, which may attend the beginning of this measure, may usually be quieted by the administration of a single dose of twenty grains of bromide of potassium taken an hour or so beforehand. Where the presence of the tube in the stomach provokes vomiting, the immediate introduction of a little fluid to remove the gastric wall from contact with the extremity of the tube will be sufficient, usually, to quiet the reflex.

The amount of fluid to be used at a time should be small at first, as vomiting is easily excited; and until the stomach has become used to the maneuver a pint will be sufficient. As treatment progresses, however, one, two or three quarts may be used at a time without inconvenience, treatment to be repeated each morning before eating.

The solutions should be warm (98.5° F. or thereabout) and may consist of simple alkaline drenches, a drachm and a half of Glauber's salts to a quart of water constituting a popular fluid for the purpose. I find weak solutions of asepsin excellent and have used boracic acid as a medicament with satisfaction. Long-standing cases of uncomplicated chronic gastritis recover completely in a few months on this treatment without the assistance of other measures except proper attention to dieting. Mucus and retained fermentative elements and products are thus removed and the mucous membrane is aroused to normal action, the hyperæmia subsiding and the irritated surface returning to a healthy condition.

The introduction of *hydrozone* as a remedy in this condition was another innovation of remarkable value. A drachm of Marchand's hydrozone added to four ounces of boiled water and drank while the stomach is empty exerts a powerful influence in dissolving and removing the tenacious mucus, destroying microbic elements of fermentation and stimulating normal action in the diseased mucous structure. The best results follow its use in the morning before breakfast, the patient taking it while in bed and remaining on the left side for ten minutes before rising. It may be taken oftener, but once a day will suffice, and it may be advantageously used in this manner after the practice of lavage.

The hydrozone may produce acrid sensations in the throat and stomach at first and the patient may complain of an unpleasant taste following its action; but as the irritated gastric surface becomes toned under its influence this will pass away and sensitiveness to its effect will subside. Where there seems to be very much objection the amount may be considerably lessened until the patient becomes accustomed to its action and until the sensitive mucous surface becomes more tolerant.

The important step in chronic gastric catarrh (as in catarrh of all other mucous cavities) is the cleansing of the part from ropy mucus, which clogs the glandular organs and serves as a nidus for the operation of agents of fermentation. Glycozone may sometimes be preferred, glycerine possessing individual virtue in certain cases of indigestion from fermentation.

With attention to such details as have already been described, little more is necessary in the treatment of this disease. Some advise, in the absence or lack of the normal amount of hydrochloric acid, that this drug be supplied, in suitable doses, well diluted with water. Benefit may sometimes follow this measure, but with the removal of morbid accumulations a normal amount of hydrochloric acid will soon be supplied by nature—all that will be required for the limited diet which the nature of the case demands. The efficiency of bitter tonics is doubtful when they are administered upon "general principles," though some of them may specifically improve the recuperative forces of the gastric mucous membrane and aid in a restoration of normal conditions. I believe *berberis aquifolium* to be one of these, its beneficial influence in catarrh of mucous membranes generally adapting it here, while it is an acknowledged stomachic of superior virtue. Ten-drop doses of a reliable fluid preparation repeated thrice daily will often assist materially in restoring a normal condition of the gastric mucous membrane and digestive glands, aiding digestion, banishing boulimia and promoting a normal appetite.

When catarrhal accumulation is a marked feature and there is a yellow coating on the tongue persistently *bichromate of potassium* in minute doses (two or three grains of the 3x) repeated three or four times daily will assist the local treatment. *Nux vomica* may relieve some of the local unpleasantness, and there are those who assert that it specifically ameliorates the catarrhal condition. The specific indications need not be referred to here, but the dose should be minute. Hydrastis, pulsatilla, robinia, antimonium crudem, bismuth (both the subnitrate and liquor) and many other remedies have their advocates.

In anæmic persons, where catarrhal tendencies are strong, *calcium*

phos. 3x in two- or three-grain doses repeated three or four times a day will lessen the ropy secretion and lessen anæmic tendencies. Protonuclein is a drug that promises much as a restorative here.

Where chronic gastritis attends malarial cachexia that group of remedies which tend toward lessening the pressure in the radicles of the portal vein will be efficient in relieving the congestion of the gastric mucosa. Of the four principal ones of these—polymnia, ceanothus, carduus and grindelia squarrossa—*grindelia squarrosa* is my favorite. Improvement in digestion under favorable circumstances almost invariably follows its use. From five to ten drops of a saturated tincture of the genuine plant administered in a swallow of water and repeated three times a day insure marked benefit within a few days. *Chionanthus* in ten-drop doses may be advantageously combined with it in most cases, especially where icteric symptoms are present.

Sometimes we may be urged to administer agents for the relief of cardiac palpitation and associate gastric distress. Cactus grandiflorus and pulsatilla possess an established reputation and they will occasionally answer us well. The best remedy I have ever tried, however, is a saturated tincture of *aploppapus laricifolius* in from two- to ten-drop doses, one or two doses at a time being sufficient for temporary relief. It calms erethism of the sympathetic nervous system, promotes rest, strengthens cardiac action, lessens pain, relieves præcordial oppression, favors evacuation of the bowels and aids digestion. Minute doses of aconite and rhus tox. are not to be despised for this condition, that reliable gastric sedative combination being very serviceable, even sometimes in chronic irritation.

Where constipation is present enemata will be found preferable to laxative medicines, the salt-water *galvanic enema* being an excellent aid in stubborn cases, it not being necessary to repeat it more than once or twice a week. The positive pole should here be applied with a moistened sponge over the epigastrium.

Local applications over the epigastrium are sometimes of excellent service and in intractable cases should be tried. The compound *tar plaster* of our forefathers, worn over the epigastrium until pustulation begins, to be removed for a few days and its use repeated again and again to perpetuate a superficial irritation, has many able advocates even at the present day; and I have known it to effect most excellent results. A *vinegar pack* or *girdle*, worn upon the epigastrium, is hardly less effective. Equal parts of vinegar and water may be employed to moisten an epigastric pad, which should be wrung as dry as possible two or three times within the twenty-four hours and worn constantly, the clothing being protected by an oiled silk covering.

As the disease is a long time in becoming established, it should be expected that several months will be required to overcome it.

Dilatation of the Stomach.

Synonym.—Gastrectasis.

Definition.—Permanent enlargement of the cavity of the stomach due to stretching of its walls, with degeneration of the muscular coat.

Etiology.—Gastrectasis may occur in an acute or chronic form, acute gastrectasis, however, being very rare. The acute form may occur as the result of the ingestion of an enormous quantity of aliment at a single meal, paralytic dilatation resulting. The usual form of dilatation occurs from causes which bring about a gradual enlargement of the stomach, the most common being pyloric stenosis. Pyloric stenosis may result from thickening of the walls of the pyloris as the result of acute or chronic inflammation, cancerous infiltration, non-malignant ulceration or fibroid induration of the pylorus. Other cases of gastrectasis may be due to atony of the muscular walls from habitual over-distention of that organ, as is common with gourmands and beer-drinkers. Paralysis of the nerve-supply, attended by impairment of normal peristalsis, is another cause of this condition, this sometimes occurring from suppurations about the stomach, such as empyæma or suppurative pericarditis; and parenchymatous degeneration, occurring as a result of scarlet fever, may impair permanently the tonicity of the gastric muscles. A somewhat rare cause of this condition may be hernia, which operates by dragging the organ downward, adhesions also sometimes acting in a similar manner. Middle-aged or elderly persons are most liable to be affected, though dilatation of the stomach may occur in children, associated with rickets.

Pathology.—The amount and character of the dilatation differ materially in different cases. Sometimes the enlargement is regular, the walls being evenly stretched so that the cardiac extremity is carried toward the left and upward, this usually being the case when there is pyloric stenosis without localized weakness of any particular part of the organ. In other cases there may be some local weakness, due to ulceration or erosion of the wall, circumscribed patches yielding to form pouches or diverticula. Stenosis is at first followed by hypertrophy of the walls of the stomach, this afterward being attended by atrophy and dilatation. The muscles may now be so thinned and stretched

Dilatation of the Stomach with Pyloric Stenosis. Dotted line represents dilatation.

as to be scarcely discernible, and fatty degeneration of its fibers may attend. Muscular atrophy is most marked where atony of the muscular wall arises independently of stenosis. Here the rugæ of the mucous membrane may have disappeared and the covering become pale and atrophied.

Symptoms.—Indigestion and gastric discomfort are the first symptoms noted. In acute dilatation there is sharp pain in the epigastric region, tenderness upon pressure and præcordial oppression with sensations of fullness. As these symptoms subside indications of chronic dilatation manifest themselves. Vomiting at intervals of enormous quantities (from one to three gallons) of food and liquid is the most usual symptom of this condition. The intervals may be two or three days in length, the material ejected usually being the major portion of what has been ingested during such periods. The vomitus consists of mucus and remnants of food, all of which has undergone decomposition, the mass exhaling a fœtid odor, presenting a frothy, yeasty appearance and being acid in reaction. Various resisting substances, such as cherry stones, grape seeds, etc., may be found. Upon microscopic examination abundance of the yeast plant and sarcina ventriculi will be found and also various bacteria. Lactic and butyric acids with various gases may be present; and hydrochloric acid may or may not be found, it sometimes existing in excess.

Pyrosis, eructations of fœtid and acrid material, heartburn with epigastric weight and pain and other gastric disturbances are almost constantly manifest. Emaciation progresses, nutrition suffering much, the skin becoming harsh and dry, the bowels constipated and the urine scanty. Muscular cramps, sometimes amounting to spasms, usually attend aggravated cases, the muscles of the calves, hands and arms being most affected. The appetite may be voracious or there may be anorexia. Usually there is a voracious appetite.

Upon inspection an eminence will be observed just above the umbilical region with a depression in the epigastrium, which may become filled after taking meals or draughts of fluid. Sometimes the stomach may be outlined by palpation, the prominence of the pylorus and gastric peristaltic action being detected. Percussion may reveal a tympanitic sound over the epigastric region when the stomach is empty and an area of dullness when it is distended, over an abnormally large space. Auscultation may reveal succussion or splashing of fluids when the abdomen is shaken, and the falling of fluids into the gastric cavity when these are swallowed.

Diagnosis.—The habitual vomiting of large quantities of decomposed food and mucus at two- or three-day intervals, with emaciation

and gastric discomfort, is sufficient to establish a diagnosis. Inspection of the material ejected may assist in doubtful cases.

Prognosis.—Therapeutic resources are usually futile in the management of such cases. Where pyloric stenosis is the perpetuating cause a cure may follow longitudinal incision and transverse stitching at the point of narrowing, this serving to widen the opening and afford a ready passage for the food. When an abstemious regimen is followed, dilatation due to simple stenosis may not interfere greatly with average longevity.

Treatment.—*Lavage*, with warm water and asepsin, is an important part of treatment. This should be repeated sufficiently often to prevent decomposition of food and avert large accumulations, thus providing against distention and weight. Instead of asepsin hydrozone may be used to medicate the cleansing fluid.

Strychnia, nux vomica, galvanism and faradism are all recommended to stimulate contraction of the relaxed muscular walls. Where the mucous membrane is atrophied *berberis aquifolium* is worthy of lengthened trial.

The *food* should be taken in small quantities and be of fluid and concentrated form. Not more than six ounces of drink should be allowed at one meal, and in bad cases no drinks at all should be allowed at meal-time, a tumblerful of hot water being taken half an hour before eating that it may pass into the duodenum and be absorbed before the food is introduced into the stomach. Fermentable foods, such as starchy articles and sugars and fruits which contain much water and vegetable acids (which are apt to disagree), should be avoided. Peptonized milk, scraped beef, lean beef free from coarse fibrin, fresh vegetables and dry bread comprise the kind of food to be taken. Some prefer a dry diet, only enough being allowed to meet the most urgent demands of the body. Fats should be discarded.

Peptic Ulcer.

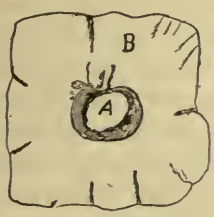
Synonyms.—Round Ulcer; Chronic Gastric Ulcer.

Definition.—An ulcer, usually single though sometimes multiple, which arises from the action of the gastric juice upon a limited region of the gastric or duodenal mucous membrane, in which nutritional disturbance has lessened the resistant capacity of the tissues involved.

Etiology.—More than twice as many cases occur among women as among men, this being due, possibly, to the fact that women are more apt to follow occupations necessitating a stooping posture whereby there is crowding of the short ribs against the pyloric extremity of

the stomach. Possibly the habit of wearing corsets and of lacing may bear somewhat upon the etiology. The most active period of life—between the ages of fourteen and thirty—is the time of greatest liability, though it may occur in the new-born babe and in the octogenarian. Anything which tends to cause thrombus of the gastric vessels predisposes to it. It is also liable to occur in chronic gastritis, hepatic and renal cirrhosis and other conditions involving obstruction of the circulation in the gastric mucous membrane. Anæmia and chlorosis are constitutional states in which there is a tendency to it. A habitual stooping posture has already been referred to as a probable predisposing cause, and it may be mentioned in this connection that it is more frequent among milliners, seamstresses, shoemakers and others whose employment calls much of the time for a bending position.

Pathology.—The ulcer occurs upon the posterior wall of the stomach near the pylorus, in a large majority of cases, though it may develop upon the anterior wall. Occasionally a peptic ulcer may develop in the duodenum and manifest the characteristics of a perforating ulcer of the stomach. Peptic ulcers vary in size from half an inch to two inches in diameter and are usually round, though they may be oblong or oval, and, when formed of several small ones, irregular in contour. They begin in the mucous membrane and excavate a sharply defined border, the opening appearing, upon autopsy, as though punched through the mucous membrane with a sharp instrument. As the ulceration grows deeper the circles grow less regular and smaller, the excavation assuming a funnel shape, until, when the peritoneum is reached, the opening may be a mere point. During the perforating process there is no active inflammatory action, though the edges of the ulcer may sometimes be thickened and indurated, while in other cases the surrounding tissues may be normal. Usually the entire mucous membrane of the stomach is involved in a chronic catarrhal condition, though sometimes the catarrh is confined to the vicinity of the ulcer.



PEPTIC ULCER.
a, perforation.
b, mucous membrane.

Perforation occurs in only about one-eighth of the cases affected with round ulcer, recuperative processes often accomplishing a restoration of the breach of continuity, a permanent cicatrix remaining. The cicatrix sometimes contracts so as to obstruct the pyloric opening or cause other deformity of the stomach.

The ulcerative process may meet with important bloodvessels in its course, and profuse hematemesis be the result of their destruction. As the ulcer approaches the peritoneal surface of the stomach

a circumscribed peritonitis is liable to be excited in the neighborhood and adhesions may take place between the part and such an adjoining viscus as the liver, pancreas, mesentery or spleen, and the fatal results of perforation thus be stayed.

Various deviations from the usual course of the ulceration may attend. The base of the ulcer may be covered with a mass of black blood, which adheres to the surface, or there may be petechial extravasations around the ulcerated space or suppuration in the coats of the stomach, with resultant phlebitis. In other cases villous growths may spring up about the base of the ulcer, upon the surrounding mucous membrane.

Though the posterior wall of the stomach is the usual location of the ulcer, it sometimes attacks the anterior wall, and perforation is much more liable to occur in this instance, as there is less probability of adhesions.

Perforation is attended by escape of some of the contents of the stomach into the peritoneal cavity, with rapidly succeeding peritonitis. If adhesions prevent the escape of the contents local peritonitis, suppuration and burrowing abscesses may follow, with fistulous openings into the pleural sac, lung, intestine, gall-bladder or other viscus.

Symptoms.—The symptoms in the beginning are often obscure. Indigestion, attended by burning, gnawing sensations, pyrosis and gastric catarrh, with jaundice and even nausea and vomiting, may be developed. Pain soon becomes a noticeable symptom and grows more and more obstinate. Though dull at first it soon becomes lancinating and attends the period of digestion, coming on soon after eating and continuing until the stomach is empty; though sometimes it does not appear until an hour or so after food is taken. After a time there develops a pain in the dorsal region, which is said to be peculiar to this disease in that it is constant, is located in the eighth or ninth dorsal vertebra, and does not come on for several months after the epigastric pain becomes established. The epigastric pain may not be severe at first, but it soon becomes excruciating during digestion, and is often relieved by change of position, pressure sometimes affording comfort—the patient finding relief by lying across a chair or with the epigastrium upon a hard pillow flat on the floor. The pain may be paroxysmal, being very severe for weeks, then disappearing for a time, with another protracted period of intense suffering. Tenderness on pressure is usually present, the patient wearing the clothing loose and objecting to anything snug about the waist. The point of tenderness is usually small, not larger than a silver dollar, and is felt on deep pressure just above the umbilicus

(in the majority of instances) and over the eighth or ninth dorsal vertebra.

Vomiting is another prominent symptom, the rejection of food occurring in a large number of cases. This occurs after the pain has become severe, the rejection of the food being usually followed by alleviation. The food is mixed with gastric juice of highly acid nature, with more or less bile, biliary material becoming quite plentiful as the disease progresses. Sometimes a patient will vomit after each meal, sometimes once a day, while in other instances two or three days may elapse between attacks.

Hæmatemesis is another symptom which occurs frequently, though not invariably. It appears in serious form after the ulceration has advanced so as to destroy the walls of arterial twigs, slight capillary hemorrhage not attracting much attention previously, the blood then passing away with the stools. When an important vessel is disintegrated, however, a large quantity of clotted blood, of bright red color, is vomited, the patient previously experiencing a sensation of faintness; and even collapse may attend, the first hemorrhage sometimes proving fatal. Repeated hemorrhages are followed by anæmia, debility and cachetic symptoms, the features becoming drawn and the skin sometimes assuming an icteric or waxy hue.

Where diarrhœa exists, as is sometimes the case, the stools are mixed with a dark, tarry material consisting of decomposed blood, to which the term "melæna" is applied.

Diagnosis.—The diagnosis is sometimes obscure. Gastralgia may be readily confounded with this disease where hemorrhage is not present, as dyspeptic symptoms, vomiting, pain and even tenderness on pressure may be present in both. Where hæmatemesis occurs there can be no possibility of an error in this respect, as it is absent in gastralgia. In cancer there is lack or absence of free hydrochloric acid in the stomach, while in peptic ulcer there is an excess of this. The epigastric tumor of cancer is absent in ulcer and pain is much aggravated by eating, while pain of cancer is seldom thus provoked. Cancer of the stomach is most apt to occur in those of middle or past-middle life, while perforating ulcer is more apt to attack younger persons. The hemorrhage of cancer also differs in character, the blood being of coffee-ground appearance, while in peptic ulcer it is bright red if the clots be broken. The cachexia of cancer is more marked than that of peptic ulcer early, and the vomiting does not always occur with immediate reference to the presence of food in the stomach. It is impossible to differentiate between a duodenal and gastric peptic ulcer during life.

Prognosis.—It is asserted by good authority that more than half the cases of peptic ulcer recover. Some terminate fatally in a

few weeks, while others may continue many months to finally recover or afterward terminate fatally. Those of feeble constitution are less apt to resist the inroads of the disease, senile subjects and delicate women being the most unfortunate victims.

Treatment.—Congestive conditions—hyperæmia—of the portal circulation should be corrected as much as possible by the use of such agents as *grindelia squarrosa*, *polymnia*, *carduus marianus* and *ceanothus*. These agents may assist in removing blood-pressure upon thrombi and restoring a normal circulation in the gastric mucous membrane.

As curative agents we must think of those remedies which exert a plastic influence upon the diseased structures. Such special remedies as *kali bichromicum* 3x, *argentum nit.* 6x and *nitrate of uranium* 3x are appropriate members of this group. *Berberis aquifolium* is an excellent remedy as in all other cases of chronic ulceration; and at the same time it is an excellent restorative of the general system, promoting digestion, assimilation and blood-making.

I have had excellent results from three-grain doses of kali bichromicum 3x repeated every four hours during the day. It certainly exerts a healing influence in such cases, and if used faithfully before too much progress has been made I believe it will cure.

The Schuessler remedies promise better results than ordinary treatment in this affection and should receive the practitioner's respectful attention in stubborn cases. The following experiences are from a paper on "Biochemistry," read before the Oregon State Medical Association September 23, 1898, by A. A. Leonard, M. D., and will apply here:

"Case III.—Miss H., aet. 19, German descent; domestic; family history good; personal history, healthy up to a year previous to consulting me, when she began to run down, had indigestion and lost flesh and strength. On previous New Year's Day (this was in March) she had vomited, she said, about a quart of blood. This, of course, was an exaggeration. Since that time she had suffered pain after eating, often vomited her meals, had acid eructations and continually lost strength. Her symptoms at the time of calling were the same, except that that morning she had thrown up a quantity of blood, and was in consequence very weak. I diagnosed gastric ulcer.

"For the three prominent symptoms—hemorrhage, acid indigestion and anæmia—I gave ferrum phos., natrum phos. and calcium phos. She had no other remedies except rest and regulated diet. There was no more hæmatemesis and after a few days I left off the ferrum phos. and continued the natrum phos. and calc. phos. for two weeks longer. The result was a surprise to me, for she gained in every way beyond my expectations. In fact, inside of two months

she was the picture of health, her appetite excellent and she was stronger and healthier in every way than she had been for several years. The cure was permanent, for I heard from her a year and a half later and she was still in good health.

"Case IV.—Miss K. D., aet. 20, American. Occupation, teacher. History of stomach-pain, occasional gnawing in stomach with some soreness for the past year. Sent for me Christmas Day, '97. I found her suffering with hæmatemesis, which was somewhat alarming. The vomiting had come on at night and had continued at intervals during the day, until she was quite weak.

"I diagnosed gastric ulcer, and to meet the first indication, the arrest of the hemorrhage, I gave her glonoin and hyoscyamine sufficient to keep the skin flushed for the first twenty-four hours. I might have stopped the hemorrhage with ferrum phos., perhaps, but hardly dared to risk it. She was put to bed with strict injunctions to stay there and remain as quiet as possible, and was allowed a tablespoonful of milk every two hours for the first day, gradually increased as the symptoms abated. After the first day I put her on natrum phos. and calc. phos., the same as Case III. She steadily improved from the first and rapidly recovered. She was kept on these remedies for about four weeks. I have recently heard from her and she has had no relapse, and is now in robust health."

Subnitrate of bismuth exerts a local influence that is worthy of consideration, though little permanent benefit can be expected from it as a rule.

Minute doses of *aconite* and *rhus. tox.* may be tried where the vomiting is intractable, and in event of the failure of this measure resort may be had to *lavage.*

In incurable cases opiates may be required to alleviate the pain. Hemorrhage should be treated by the recumbent posture and the administration of ten-drop doses of *erigeron canadense,* hypodermic injections of *ergotine* in two- or three-grain doses or other astringents.

The patient should remain quiet in bed to insure *rest* and a recumbent position, and the *diet* should be liquid, bland in character and should be administered in small quantities, in order to avoid distension of the stomach and risk of perforation. After hæmatemesis the stomach should be allowed to remain quiet for a time, and food should be introduced into the rectum in the form of nutrient enemata. When food is taken into the stomach, a milk diet is appropriate, though care must be observed that it be not taken so as to result in the formation of firm curds. On this account Horlick's malted milk may be preferable, though the addition of a tablespoonful of lime-water to a pint of raw milk will provide against this to considerable extent. Where acceptable, butter-milk or koumiss will

be appropriate for nourishment, and all danger of curds will be avoided. Almost any form of liquid diet which will not irritate delicate structures will be proper, and rotation among several kinds will encourage the patient to take sufficient for sustenance. After hemorrhage the quantity taken at a time should be limited to a few teaspoonfuls, larger and larger quantities being given gradually until three or four ounces are administered at a time as bleeding ceases.

Milk, beef-juice, broths, malt extracts and other fluid foods may be allowed until convalescence is announced, when scraped beef, chicken, fresh sweetbread, tapioca and rice pudding, etc., may be allowed in small quantities, the patient remaining quiet in bed for another month, in order to allow the new structure to acquire appropriate strength. A recent addition to the materia medica, and at the same time an excellent nutrient, is preserved beef's blood—*bovinine*. Thirty drops of this in a cup of hot water, repeated every three or four hours, may afford good results.

Cancer of the Stomach.

Etiology.—Age and location are probably prominent among the etiological factors of gastric cancer. As in cancer in other locations, those past middle life are most liable to this kind, about one-third of all cases of primary cancer having their origin in the stomach. Osler states that cancer of the stomach is only second in frequency to that of uterine cancer. It is more frequent in males than females, in the ratio of about five to four. Local irritation, doubtless, contributes to the predisposition of this part to malignant disease, the almost constant disturbance which it undergoes as the active organ in receiving and reducing food for digestion probably being contributory, while accidental irritation from indigestible and acrid materials which are swallowed, and the pernicious habit of prescribing irritating cathartics so fashionable among a large class of physicians, assists in contributing to the cancerous tendency. Long-continued irritation of such kind may finally give rise to the development of the new growth, the disease being rare in children and uncommon before the age of forty.

The favorite seat of gastric cancer is the pylorus and when located here the upper portion of the duodenum also is usually involved. The next point in frequency of attack is the cardiac extremity and lesser curvature, the lower portion of the œsophagus then usually being implicated. Cancer of the stomach is a common disease in this country, and should be suspected in all cases of gastric trouble attended by rapidly encroaching debility and emaciation.

Family tendency is somewhat marked, something like one-seventh

of the cases occurring probably belonging to this class. It is considered, by good authority, doubttul that depressing emotions, mode of life or previous disease exert any influence in the etiology of the affection. Various popular beliefs exist that cancer is due to the influence of certain foods. For example, many believe that the eating of tomatoes predisposes to cancer, a proposition which seems ridiculous from a scientific point of view.

Pathology.—While the pylorus is the most frequent seat of cancer and the cardiac extremity next, the curvatures, fundus and body may all be affected by the primary growth. There is a difference of opinion among medical authors as to which form is most common, some asserting that scirrhous cancer is most frequent and others claiming the supremacy for epithelioid. My own experience leads me to favor the opinion that scirrhous is most often found, although I have no statistics to offer. Epithelioma occurs frequently, and all forms may become colloid or gelatiniform in character during their progress. In many instances the morbid growth may be a combination of several varieties.

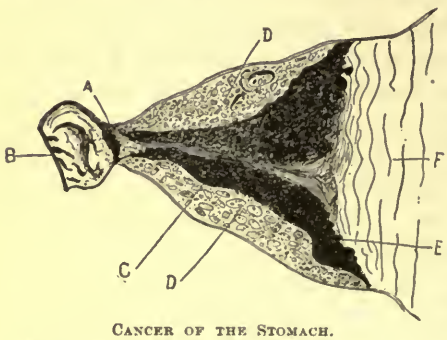
Scirrhosis of the stomach develops in the submucous structure, small, grayish nodules enveloping the extremities of off-shoots of the gastric tubules which have pushed their way into the submucous tissue, the character of the growth then being, in reality, epithelial, though the fibrous stroma is greatly in excess of the cell-element. As the disease progresses the fibrous structure encroaches upon the mucous membrane, puckering it into nodules and pushing them outward into the cavity of the stomach in polypoid forms, rapid increase of new tissue thickening the pyloric wall and narrowing the lumen, the growth extending along the greater and lesser curvatures toward the dilated portion of the stomach. The muscular and areolar layers become fused into an indistinguishable mass after a time, the surface of a fresh cut presenting a whitish, glistening appearance, with pearly settings, the individuality of the mucous, submucous and muscular layers being entirely lost. As the pylorus becomes occluded the unaffected portion of the stomach becomes dilated, though sometimes its walls are shriveled and contracted with leathery thickening of the entire structure. Chronic gastritis may arise from pressure of the indurated part against the unaffected mucous membrane and the usual appearance of such condition may be manifested beyond the cancerous mass. As ulceration develops there is liability to perforation of the gastric wall, the opening sometimes entering the peritoneal cavity, sometimes penetrating the duodenum or other neighboring organ and sometimes even forming an external opening through the anterior wall of the abdomen. Secondary cancerous deposits are common, the liver being most frequently involved, then

the lymphatic glands and neighboring intestines, especially the rectum. The kidneys, pancreas, spleen, bladder and other abdominal and pelvic organs, as well as those of the thorax, are liable to be secondarily involved.

Encephaloid cancer begins in the submucous tissue, though the nodules are much softer, the stroma being less abundant and the cells more numerous. It develops more rapidly than scirrhus and is more vascular, large fleshy spongy excrescenses projecting into the gastric cavity.

Colloid cancer of the stomach is rare. It is said to begin in the glandular structure of the gastric wall, though t invades all the coats with great rapidity and also involves neighboring organs in the same manner. It does not appear in the form of nodules but as an irregular mass of gummy, glistening material inclosed in large alveoli. The entire structure of the stomach is much thickened, and on the inner surface there are closely-set cavities of honey-comb appearance marking empty alveoli, which have discharged their contents.

Symptoms.—Rapid loss of flesh and strength with dyspeptic symptoms is sufficient to warrant suspicion of cancer of the stomach. Sometimes the gastric symptoms are more marked and there will be anorexia, nausea and vomiting and pain after eating. Anæmia of a peculiar character soon develops, the skin presenting a peculiar sallow, clayey or waxy appearance and being leathery and inelastic to the feel. The pulse is increased in frequency and becomes small and feeble, and these symptoms are steadily aggravated in spite of treatment. In colloid cancer, where the entire stomach is sometimes involved, there may be tumultuous peristaltic action of that organ at times, though this is not common. Where the pylorus is mostly affected a tumor may soon be felt just above the umbilicus, which is hard, firm and immovable and which usually pulsates from the impact of the abdominal aorta. When the cardiac extremity is the part involved, the tumor is not perceptible upon palpation.



CANCER OF THE STOMACH.

a, pylorus.
b, duodenum.
c, passage through cancerous growth.
d, cut surface of cancer.
e, diseased mucous membrane.
f, normal mucous membrane wrinkled transversely from cancerous contraction.

Dyspeptic symptoms are so common in other cases that they are not highly suggestive of cancer, and as cancer patients are liable to be dyspeptic subjects long before the malignant disease develops,

these do not attract much attention until they become extreme. Vomiting, however, soon draws attention to the gravity of the case. This may occur only occasionally at first, perhaps not oftener than once every three or four days, but it usually increases rapidly in frequency and after a few weeks may recur several times a day. It is most apt to be severe when the malignant growth is about the orifices, vomiting occurring soon after eating when the cardiac orifice is affected and after a considerable interval where the growth is about the pylorus. The vomiting consists of food and mucus mixed with various acids and exhaling a sour and fœtid odor. After ulceration of the morbid growth begins hæmatemesis is not uncommon, the blood being mixed with other material or so altered by the secretions as to present a dark brown or black appearance, then termed "coffee-ground" vomit. The yeast plant, various bacteria and sarcinæ ventriculi are present, though not so common as in gastrectasis.

Much stress is placed by many diagnosticians upon the absence of free hydrochloric acid from the gastric secretions. To determine this administer a test meal, consisting of a breakfast of a Vienna roll with a cup of tea without sugar or milk, and after an hour remove some of the contents with a stomach-tube for examination. The method of Günsburg for detecting hydrochloric acid is simple and effective: Mix phloroglucin two parts, vanillin one part and alcohol thirty parts. Add a drop of this to a drop of the gastric contents (filtered) on a porcelain plate and evaporate to dryness, watching the reaction. If free hydrochloric or other mineral acid be present a handsome rose-red color begins to appear at the edges. As hydrochloric acid may be present under other circumstances as, for instance, when there is atrophy of the gastric tubules, this symptom cannot be considered diagnostic and may only be taken as corroborative when other indications of cancer attend.

Pain is a common symptom of gastric cancer, though quite a number of cases run their course without—unless it be that which attends peritonitis after perforation has occurred. The pain varies in its situation, though it is most commonly in the epigastrium. Sometimes it is almost confined in the dorsal region, sometimes it is felt most under the scapulæ, and occasionly it lingers about the loins. It is burning and dragging in character, hardly ever being lancinating or excruciating, as in peptic ulcer.

Dropsical symptoms are likely to appear during the advanced stage of gastric ulcer. They are first observed about the ankles and legs, these becoming swollen and œdematous, especially after the patient has been upon his feet for a few hours in the morning. As further progress is made, ascites or anasarca may arise.

The wasting of flesh affects the heart muscle and its debility is

marked during the last stages of the disease, the pulse being weak and rapid; and it may seem, from the acceleration of the pulse and hot skin, that febrile symptoms are present. Indeed, this may sometimes be the case, chills and fever, with elevation of temperature to 102°—104° F. (followed by profuse sweats) arising temporarily, though the temperature is usually normal or subnormal. The febrile paroxysms are probably due to suppurative action and are not common among the early developments of the disease.

As secondary cancer involves other organs a marked modification of the symptoms may result. Cancer of the liver may give rise to jaundice, with enlargement, pain and tenderness in the right hypochondrium. Ascites may now be a direct result of this condition through obstruction of the portal circulation. Extension of the cancerous infiltration to the peritoneum may give rise to widely diffused pain over the abdominal region, and obstruction of the vena cava ascendens results in dilatation of the superficial epigastric and other subcutaneous abdominal veins.

Diagnosis.—In ordinary cases, especially where the pylorus is the seat of the affection, the diagnosis is comparatively easy. The gastric disturbance with vomiting, the rapid loss of flesh and strength, the epigastric tumor readily felt through the attenuated abdominal wall as a hard, immovable body (pulsating with the aortic impact), with the constant, burning pain, can hardly be mistaken for symptoms of any other disease. In other cases, however, the tumor may be so located that it cannot be discovered by palpation, the gastric disturbance may be slight and the constitutional symptoms may readily be taken for those of pernicious anæmia. In other cases the constitutional symptoms may not appear prominently until near the last and the local symptoms may be mistaken for those of chronic gastritis or gastrectasis—until the "coffee-ground" vomit appears. Secondary affection of adjacent organs early may also obscure the diagnosis.

Prognosis.—Invariably unfavorable. The most that can be promised is temporary palliation of the pain and other unpleasant symptoms. The average duration of cancer is two years, though as the disease will have progressed considerably before a diagnosis can be made it is estimated that few survive more than a year after that time. Some cases run a rapid course, a fatal termination being reached in from three to six months. A cure is barely possible.

Treatment.—In pyloric cancer with stenosis *lavage* may afford some relief from the gastric unpleasantness by neutralizing accumulated fermentative products and removing superfluous mucus. *Hydrozone* or *asepsin* may be employed for this purpose as directed under

chronic gastritis. As there is some danger of perforating the weakened wall of the stomach, discretion should be observed in the use of the stomach tube, hydrozone alone sufficing to remove the mucus without lavage. *Cundurango* assists in relieving the pain and vomiting in some cases and is always worthy of trial. *Echinacea* is almost a specific in the pain of cancer and should be tried here early, though it may not be reliable to quiet the vomiting. *Bovinine* is another agent that is worthy of trial, as its nutrient qualities are combined with excellent anodyne and calmative properties. Some cases may justify abdominal section and resection of the pylorus, though a fatal result may be expected within a few days in most instances. The use of opiates freely is fully justifiable during the last stage, though echinacea is more reliable as a pain reliever.

One or two drops of *carbolic acid* mixed with glycerine and diluted with water will often prove exceptionally valuable in controlling the vomiting.

Chelidonium has recently promised much—curatively—in this disease and it will probably act best in combination with echinacea. The dose may vary from fifteen to twenty drops of the specific medicine. Another measure promising much is that of Cutter—a strict diet of chopped beef and hot water, without tea, coffee or milk.

A still later acquisition to the list of curatives in cancer is *eucalyptus*, which might be tried here, an approximate dose being ten drops of a saturated tincture of the fresh leaves.

The *dietetic* treatment of cancer of the stomach is very important. Solid food, after the disease has progressed appreciably, should be discarded altogether for fear of perforation or hemorrhage. Even such liquids as require stomach digestion ought to be largely avoided and reliance for nourishment had upon liquid predigested food, such as beef peptonoids or pancreatinized or peptonized milk. However, as these may become distasteful after protracted use without variation, rice-water and various vegetable and animal soups may be alternated to vary the routine. If chopped-beef diet is to be employed it should be begun early and the beef should be minced exceedingly fine. Black tea and coffee without milk may be allowed sparingly. If the patient desires a small amount of claret or port wine may be taken, unless it provokes gastric discomfort.

Non-malignant Tumors of the Stomach.

These growths are seldom found, and when present are small and cause little trouble. Polypi are the most common variety. They are usually multiple and may be very numerous, as many as a hundred and twenty having been reported in a single case. They

consist of hypertrophied mucous follicles, which become pedunculated. Fibromata and lipomata are seldom met with. Lymphomata may develop during the progress of leukæmia. Foreign bodies in the stomach may he mistaken for tumors when palpation alone is relied upon.

Hæmatemesis.

Synonym.—Gastorrhagia.

Etiology.—Many causes conspire to bring about vomiting of blood, though the most common one is rupture of the bloodvessels of the stomach. The capillaries of the vessels are most frequently ruptured, though bleeding often occurs from rupture of branches of the gastric artery. The exciting causes may be divided into:

Traumatic, produced mechanically—from perforation by a stomach-tube, by hard food, by irritant chemicals or by external violence.

Diseases of the blood, as occurs in such infectious diseases as yellow fever, malaria, typhoid and typhus, etc.

Congestion of the gastric mucous membrane, as occurs in acute gastritis and vicarious menstruation; in active congestion and the passive congestion which attends obstruction of the portal circulation, as in hepatic cirrhosis, thrombosis of the portal vein and other causes of retardation of the flow of blood through the liver or along the branches of the portal vein, such as pressure from abdominal tumors, etc. Hepatic or pulmonary diseases obstructing the return of venous blood to the right auricle may also operate in this direction through backward pressure. Similar effects may arise from omental hernia when this exerts a dragging influence upon the stomach.

Local diseases of the stomach may be attended by ulcerative abrasion of the gastric vessels, as in ulceration from chronic catarrh, peptic ulcer or cancer. Varicose conditions of the gastric veins or aneurism of a branch of the gastric or splenic artery may result in rupture, or such an accident may occur during violent retching or vomiting.

Nervous conditions, such as progressive paralysis of the insane, epilepsy or hysteria, the character of the accident then sometimes being inexplicable. Sometimes the blood may be swallowed as a result of bleeding in the pharynx, nasal passages, larynx or œsophagus and afterward vomited. Malingerers and hysterical persons sometimes swallow the blood of animals and afterward vomit it up for mercenary purposes or to excite sympathy.

Pathology.—A variety of conditions may be found after death from hæmatemesis. In hæmatemesis from cirrhosis of the liver no

local lesion can be found, the blood having probably passed into the stomach by diapedesis from the gastric capillaries. Or there may be a rupture of a submucous vein and the erosion of the mucous membrane escape notice on account of the minute size and post-mortem changes. Miliary aneurisms may communicate with the cavity of the stomach by pin-hole perforations and post-mortem appearance afford no explanation of the morbid condition. When the hemorrhage results from portal obstruction no lesion is observable, except that the mucous membrane is smooth and pale in appearance. Intestinal ulcers tell their own stories.

Symptoms.—Hemorrhage and later anæmia are the prominent symptoms. There are cases, however, where neither of these symptoms is manifest, either because the quantity of blood is so small that it does not cause emesis or because the amount is so large that immediately fatal results occur before it becomes developed. The blood may be ejected by regurgitation or by severe vomiting and by all grades between. The amount may vary from a few streaks in the vomited material to as much as three or four pounds in twenty-four hours. In nearly all cases a portion of the blood may pass into the intestines and in some cases the entire amount will be discharged this way, the stools then presenting the dark tarry appearance observed in hemorrhage from the upper intestinal canal. The secretions of the stomach destroy the fresh appearance of blood in the stomach after a little while, and when it remains in the stomach for a while it is dark and grumous (like coffee-grounds) and the clots, if any appear, are dark and irregular when vomited; but if ejected soon after leaving the bloodvessels its appearance is little altered. The anæmia varies in degree, according to the amount of blood lost.

Diagnosis.—Care must be observed not to confound hæmatemesis with hæmoptysis. Attention to the following points will afford valuable assistance in this direction: In hæmatemesis the blood is expelled by vomiting and if there be any cough present it occurs after vomiting, while in hæmoptysis the cough occurs in the beginning and if vomiting comes on afterward it is excited by the coughing. In hæmatemesis the blood is liable to be mixed with particles of food, while in hæmoptysis the blood is clear and frothy and râles may afterward be heard over the lungs. In hæmatemesis the blood is expelled in quantities with complete intervals, while in hæmoptysis there is repeated and frequent expectoration of blood with cough after the principal amount has been expelled. Physical examination should be made in all cases where there is any doubt, auscultation of the chest assisting in determining any question in the matter.

Prognosis.—Fatal result may attend aneurism or the rupture of a large vein in the gastric walls, but other gastric hemorrhages

are seldom fatal. Hemorrhages from cirrhosis of the liver or other portal obstruction are more dangerous than those from ulcer or cancer.

Treatment.—Absolute rest in the recumbent position is an important element of treatment. All food by the mouth should be discontinued, the patient being supported by rectal alimentation, and thirst should be relieved by sucking small pieces of ice, drinking being avoided. *Ergotin* and morphia may be given hypodermically to constringe the bloodvessels and promote rest from peristalsis. In extreme cases brandy per rectum will assist against collapse. After the active symptoms subside food should be liquid in form and only small quantities should be taken at a time until eroded vessels have had time to heal. When portal obstruction is the cause of the hemorrhage, efforts should be made to relieve the condition, fullness of the abdominal capillaries being restrained in some measure by such remedies as polymnia, ceanothus and carduus marianus. In malarial hæmatemesis grindelia squarrosa would be preferable. Ulceration of the stomach should be met by appropriate treatment.

Functional Gastric Dyspepsia.

Synonym.—Nervous Dyspepsia.

Definition.—Indigestion in which there is no observable organic lesion to account for the disturbance.

Etiology.—The causes of this form of indigestion are numerous, and usually of reflex character. Nervous dyspepsia is a common accompaniment of uterine lacerations and almost a constant symptom—in greater or less degree—of such rectal irritation as that attending hemorrhoids, rectal pockets and papillæ. It may be brought on by severe mental occupation associated with sedentary habits or by depressing influences continued for a lengthened period. Rheumatic affection of the muscular walls of the stomach may be attended by difficult and painful digestion, it then being termed gastralgia or gastrodynia. Impairment of the functions of the secreting glands of the stomach may also arise, subacidity or hyperacidity being the condition. Anæmia and neurasthænia are commonly attended by functional dyspepsia, and malaria may be an important factor.

Pathology.—Careful examination will determine the absence of any structural disease; though digestion is attended by various kinds of discomfort there is lack of any local structural change to account for it. Often the irritation is at a remote distance from the stomach, and so far as its structural character is concerned of a trifling nature, but such as to constantly tease the terminals of sympathetic nerves.

Symptoms.—The symptoms will vary according to the special character of the affection, though there is much in common with them all. Pain of burning character often attends, though instead of scalding sensations in the stomach there may be sharp, lancinating distress of neuralgic character. Sometimes there is aching in the epigastric region with sensation as though there was a hardened ball in the stomach, and again the painful sensation may be that as of a gnawing in the part. An unpleasant sensation of fullness in the epigastric region follows eating in most cases, accompanied by præcordial distress and dyspnœa. Eructations of food and acid material are common, and peristaltic unrest, attended by gurgling, borborygmus and abdominal pain, is a frequent symptom. In many instances there is pectoral and cardiac pain during digestion. Irritability of temper and melancholia commonly attend. Vomiting attends some cases, the food being ejected from the stomach soon after meals.

Diagnosis.—The diagnosis of nervous dyspepsia from organic affections of the stomach is not always easy. In functional dyspepsia, however, it should be recollected that though much distress may attend the process of digestion, it is completed during the physiological time-limit. Seven hours after the ingestion of food the stomach should be found empty. A careful inspection of the contents of the stomach during digestion may throw much light upon the character of the affection as regards hyperacidity, subacidity, etc.

Prognosis.—Every case of functional dyspepsia ought to be cured, as there is usually a removable cause, and it remains for the physician to search this out and correct it.

Treatment.—The practitioner should inquire carefully into the habits of his patient to determine whether or not the condition depends upon some indiscretion of diet, mental taxation, sexual excess or abuse or other avoidable cause. If such exist a cure depends much upon a radical and permanent reform in this direction. The diet should be carefully selected with reference to the avoidance of articles that are known to disagree; fats, starches, sugars, tea and coffee being avoided, and the general health attended to by judicious application of exercise, genial companionship and pleasant surroundings. Malarious conditions attended by dyspepsia will demand the employment of *grindelia squarrosa, polymnia, chionanthus* or *carduus marianus;* and constipation must be properly treated, the use of copious *enemata* of salt water being efficacious for this purpose, daily irrigation of the rectum and colon being sufficient to afford relief. A careful examination of the orifices of the body should be instituted to determine their condition and enable the practitioner to decide as to the necessity of surgical interference. Lacerated cervix, rectal pockets, papillæ, hemorrhoids, etc., should be suspected (if not inducing the condition)

whenever present. The vomiting of food, which attends some cases of the kind, will almost always disappear upon the removal of rectal or uterine irritation, of which gastric symptoms are but reflexes.

Sometimes functional dyspepsia depends upon the presence of prostatic irritation and demands the judicious employment of *galvanism*, with the aid of such sexual tonics as *saw palmetto*, *salix nigra* and *viburnum*.

Berberis aquifolium and *piper methysticum* are especially valuable as encouragers of the digestive functions. The dose of either may vary from ten to fifteen drops of the specific medicine or some other reliable preparation administered in a little water before each meal and at bedtime.

Self-*massage* immediately upon rising in the morning is an excellent practice for nervous dyspeptics. All the muscles of the body should be thoroughly rubbed and kneaded, to be afterward well pounded with the fists, the epigastric region receiving special attention of this kind. The effect of such treatment upon all the organs of digestion is excellent, and superior to that of drugs alone.

Præcordial oppression yields to small doses of *aploppapus lar.*, one or two being taken soon after eating (five or ten drops of the tincture) an hour or more apart.

Painful conditions of the stomach may require the use of *caulophyllin* (3x trituration), a decoction of *cimicifuga*, specific *colocynth*, *dioscorea* or other especially demanded drug.

Hypersecretion and Hyperacidity.

Normally, the gastric juice is secreted only during the process of digestion. When its secretion occurs between such times, it constitutes hypersecretion. It is usually associated with some nervous disorder, such as locomotor ataxia or neurasthenia. Hyperacidity is a more common condition and is an aggravating attendant of gastric ulcer.

Symptoms.—There is burning, gnawing pain in the stomach and substernal region with acid eructations which set the teeth on edge. In aggravated cases vomiting of gastric juice may occur. All these symptoms are ameliorated by eating. The eructations and sour risings of chronic gastritis, dilatation, etc., occur after eating, while in this case they occur during fasting, and there is no admixture of food in the eructations as in those cases. Starchy food is digested slowly.

Diagnosis.—A test-breakfast of a Vienna roll and a cup of tea without milk or sugar may be ordered and an hour afterward some of the contents removed through a stomach-tube may be subjected to

quantitive analysis to determine the question of hyperacidity. If there be hypersecretion the presence of hydrochloric acid in the stomach during fasting hours will determine the matter.

Treatment.—In hypersecretion *lavage* with weak alkaline solutions should be practiced every day, the irrigation to occur before the principal meal. Minute doses of *jaborandi*, *hydrastis* or *menispermum canadense* should be administered for their tonic influence upon the gastric tubules. Where hyperacidity is present an alkaline treatment should be administered, and a diet consisting principally of proteids, such as lean meat, largely adhered to, the patient also taking plenty of out-door exercise and occupying the mind with active diversion. *Berberis aquifolium* is an excellent remedy where there is excessive activity of the gastric tubules, the dose ranging from five to ten drops of a reliable fluid extract three or four times daily. *Cactus grandiflorus* is another remedy which sometimes proves useful in such cases, the dose being from three to five drops three or four times daily.

Gastralgia.

Synonym.—Gastrodynia.

Definition.—Pain in the stomach without organic disease or disturbance of digestion.

Etiology.—Gastralgia is due to a variety of causes. It may be neuralgic or muscular, many cases being due to the fact that the subject is prone to attacks of muscular rheumatism, the disease frequently involving the muscular walls of the stomach. In other cases it may occur as the leading symptom of an attack of malaria, the pain coming on periodically and being amenable to the curative action of antiperiodics. In other cases it may be purely reflex, depending upon uterine irritation, such as laceration of the cervix uteri, in which case it is likely to appear coincidentally with the menstrual period. Or, rectal irritation may give rise to it through reflex action, aggravation of the local irritation being transferred to the stomach instead of being appreciated at the actual point of disturbance. Some persons of gouty tendency are liable to such attacks, and lead poisoning may manifest itself in severe paroxysms of gastrodynia. The abuse of narcotics, such as tobacco and tea, may lead to attacks of this kind.

Symptoms.—Sudden and excruciating pain in the epigastric region is the initial symptom in the majority of cases, though there may be premonitory loss of appetite, nausea and other gastric unpleasantness for a few hours prior to the attack. The pain may be burning, boring, griping or aching in character and it may radiate

to the back and around the waist, the pain being constant or intermittent. Eating sometimes relieves and hardly ever aggravates, unless it be in gastralgia of rheumatic type, when eating may provoke aggravation. Pressure may aggravate if firm, though light pressure may afford comfort. Subjects of gastralgia are usually persons of highly nervous temperament.

Diagnosis.—This consists of the exclusion of organic disease.

Prognosis.—The cause is usually detectable by an intelligent and properly educated physician, and its removal will insure recovery.

Treatment.—The first demand is for temporary relief from an attack. Evacuation of the bowels with enemata and the application over the epigastrium of a folded handkerchief moistened with *chloroform* and covered with a towel wrung out of hot water will usually afford relief, though an internal administration of morphine may sometimes be required. Darting, radiating pains sometimes yield to *piper methysticum* and sometimes to *colocynth*, *dioscorea* or *bryonia*. Copious draughts of hot water sometimes afford relief. A lacerated uterus should be repaired and rectal pockets, ulcers, papillæ, hemorrhoids and spasmodic stricture should be properly treated when present in any persistent case. Periodical attacks may require antiperiodic doses of *quinine* followed by the prolonged administration of *grindelia squarrosa* in appropriate doses. If lead poisoning be suspected the proper remedies for this condition should be prescribed. In persistent cases a radical change of climate may succeed in relieving when other treatment fails.

Many cases of gastralgia are rheumatoid in character and occur in persons subject to muscular rheumatism. In these cases we will get prompt results in most instances from generous doses of *cimicifuga*. A strong decoction of the recently dried root in wine-glassful doses repeated every few hours will usually relieve acute cases within a short time. Smaller doses continued for a few days at longer intervals will usually confirm the cure. In malarious regions its action, however, may need to be supplemented by that of a properly selected antimalarial agent. In other cases, especially those which have reached a more chronic stage, minute doses of *caulophyllin* may be more efficacious and at the same time more acceptable. Two or three grains of the 2x or 3x trituration of a good article of caulophyllin should then be administered every three or four hours during the day for a week or more. Sometimes *rhamnus californica* will serve a better purpose and afford more prompt relief. A tablespoonful of a strong decoction of the bark may be administered every two hours until a laxative effect becomes manifest, after which the dose should be lessened or the remedy discontinued altogether.

Nervous Vomiting.

This sometimes occurs when there is no organic disease to cause it, the patient being unable to retain food of the simplest character in the stomach. It is usually reflex in character, the vomiting of pregnancy and sea-sickness being familiar examples. In other cases, however, the cause may be more obscure and the condition may be so persistent as to threaten inanition. In one case of this kind, where the patient had been consigned to death by her physicians under the diagnosis of cancer of the stomach, I afforded prompt and permanent relief by simply stretching the sphincter ani with the thumbs. Doubtless most of these cases may be referred to reflex rectal irritation, though some may be due to uterine irritation or other reflexes. In managing them the physician should examine for all possible sources of reflex disturbance, and when a probable cause is found it should be corrected at once. If this fail further investition should be pursued.

Peristaltic Unrest.

Some persons, more especially women, are subject to loud gurgling or splashing sounds in the epigastric or left hypochondriac region, due to peristaltic unrest of the stomach. This is probably a reflex due to some distant disturbance and is not incompatible with average health. General tonic faradic treatment with local faradization of the affected region is beneficial, though permanent relief is not always accomplished.

Rumination.

Hysterical and feeble-minded persons sometimes regurgitate and chew the food like cud-chewing animals. It is a disgusting practice and difficult to cure, though there is little danger of evil effects from it.

VII. DISEASES OF THE INTESTINES.

Morning Diarrhœa.

This is a functional affection, the etiology and pathology of which are obscure. It consists of a diarrhœa, which comes on during the early morning hours, sometimes rousing the subject from slumber, the evacuation being usually preceded by more or less severe griping pain in the abdomen. In many cases one evacuation ends the trouble for the day, though several may follow, the diarrhœa extending well into the forenoon.

It is possibly due to mental disturbance, such as worry or overwork, and is apt to be aggravated by late hours with evening lunching. Irritation of the defecatory center may arise from prolonged riding over rough roads, as is the lot of many country doctors who suffer from jar of the spine. Rectal irritation may be a cause through reflex action. Sometimes the trouble may cease for weeks or months, to return upon deterioration of the general health.

Treatment.—Late hours of dissipation should be carefully avoided and a very light supper, if any, should be taken, and this not after six P. M. Mental strain should be prevented, the patient taking exercise on foot in moderation but refraining from riding or any exercise which incurs jarring of the spine. Careful inspection of the rectum should be made to detect and correct any local lesions, if any exist. Massage of the abdomen and faradization of the entire abdominal surface with the negative pole of a faradic battery—the positive pole being held at the nape of the neck—should be repeated twice a week. Minute doses of *nux vomica* (one drop of the tincture in a glass of water before each meal) may be tried with some assurance of benefit. A radical change of climate will promote the best results.

Acute Intestinal Catarrh.

Synonyms.—Acute Catarrhal Enteritis; Acute Entero-colitis; Acute Diarrhœa.

Definition.—An inflammation of the intestinal canal involving the small intestine and in many cases the upper portion of the large bowel, attended by catarrhal symptoms signalized by mucous diarrhœa with griping pains.

Etiology.—The causes have been divided into primary and secondary.

Primary causes may be (1) toxic or irritating foods or drugs of alkaline, acid or corrosive nature. (2) Errors in food either in quantity or quality, some articles of diet proving especially detrimental to certain individuals. (3) Impure drinking-water may contain elements which give rise to intestinal irritation and inflammation. (4) Certain changes in the intestinal secretions are believed to result in enteritis. (5) Sudden changes in temperature, resulting in congestion of the mucous membrane, similar to that which causes "colds" to center upon the pulmonary mucous membrane.

Secondary causes are: (1) The irritation resulting from some of the infectious diseases, such as typhoid fever. (2) Certain cachetic conditions, such as tuberculosis, Bright's disease, cancer, anæmia, etc., may be complicated by acute attacks of enteritis. (3) Chronic

congestion of the portal circulation. (4) Peritonitis, cancer of the intestines, intestinal ulcer, hernia, etc.

Among the toxic or irritating articles of food may be mentioned the toxines developed by the decomposition of cheese and milk. Arsenic, mercury and other mineral substances act in a similar way. Unripe fruit, green corn and other such materials are common causes of the disease during the heated months of summer and early autumn. Excessive secretion of bile is supposed to be one of the causes of this disease, and mental emotions may give rise to such a disturbance of the intestinal mucous membrane as to provoke irritative diarrhœa.

Pathology—The branches of the mesenteric artery distributed to the intestinal mucous membrane are injected and swollen, the entire mucous membrane is reddened and engorged and the surface is covered with an excessive amount of mucus. The solitary and agminated glands are enlarged and stand prominently out upon the mucous membrane and ulceration of the follicles and mucous surface may occur. After death the reddened mucous membrane becomes grayish, sodden and flabby.

Symptoms.—Colicky pains announce the advent of the disease the pains preceding the evacuations for a few seconds, the call to stool being urgent and the evacuations forceful and, if the lower bowel is involved, marked by rectal tenesmus. More or less tympanites and borborygmus occur during the attack, these usually being most marked shortly prior to evacuating. There are loss of appetite, nausea, thirst, dryness of the tongue and sense of debility and prostration. The skin is dry and harsh in some cases, though a relaxed sweat may attend the demand for evacuation. Slight fever may appear, though the temperature is more commonly normal. The evacuations vary, their condition depending upon the period of the disease, the portion of the intestine most involved and the amount of ingested food in the canal. They are thin and gruel-like in consistency and vary in color in proportion to the amount of bile contained in them. They may contain portions of undigested food (lienteria) and much mucus, especially when the colon is affected. From five to twenty evacuations may occur in a day.

Treatment.—The recumbent position is to be strictly maintained throughout the progress of the disease, as frequent rising to stool proves a continual aggravation. The therapeutic effect of the best remedies may be entirely lost if the patient is not kept down. A bed-pan is to be used and the patient enjoined to make as little exertion as possible. *Aconite* and *ipecac*, in combination, is the ideal prescription where a large portion of the intestine is involved. The dose should be small and frequently repeated, and relief of perma-

nent character will follow within a few hours—at least within a day or two. The following combination represents the proper proportions: ℞ Lloyd's or Worden's aconite gtt. v–x, specific or normal tincture ipecac gtt. x–xx, water ʒiv. M., and order a teaspoonful every hour.

Where the upper portion of the intestine is principally involved, *kali bichrom.* 2x or *arsenicum* 3x may be more efficacious. Two grains may be administered every two hours. The 2x trituration of *podophyllin* sometimes acts especially well here also, the same directions as to dose and frequency of administration being observed.

Rhus aromatica acts better than any of these remedies during some attacks, especially where provoked by hot weather and fermentative processes. From two to ten drops of the specific medicine may be administered every hour and repeated until benefit follows, then every two hours until complete relief is obtained.

Where the lower intestine is the part principally involved *merrius cor.* 6x may be expected to yield better results than any of the remedies already named. Five or ten drops of the dilution or two or three drops of the trituration may be administered every two hours until the disease is controlled.

Colocynth should take preference of all other remedies where pain is the prominent symptom. It is especially indicated where there is intense griping pain about the umbilicus accompanied by spasmodic cramps of the abdominal muscles just prior to the evacuations. Half a teaspoonful of the 2x or 3x dilution may be added to half a tumblerful of water and a teaspoonful administered every half-hour or hour until relief follows. It may be necessary to follow relief from pain with one of the remedies already named to complete a cure, though this will not always be necessary, colocynth frequently being curative as well as palliative.

Where typhoid symptoms appear and the patient is delirious, with dry, reddened tongue, nausea and vomiting, *rhus tox.* should be combined with the properly selected remedy. Where the discharges are dark and offensive, suggesting putrid conditions of the intestinal mucous membrane, *echinacea, baptisia* or *sulpho-carbolate of sodium* may be required. The sodium salt may be administered in quarter-grain doses repeated every three hours.

Arseniate of copper 3x is strongly indicated where the evacuations are watery and are voided with a forcible gush. One-grain doses of the trituration may be administered at one- or two-hour intervals. Small doses of the 3x dilution of *veratrum album* answer as well if not better in some cases, and sometimes wineglassful doses of a decoction of the fresh plant *erigeron can.* repeated every half-hour or hour may succeed better.

Some allopathic practitioners advise the administration of cathartic doses of castor oil or calomel in the beginning, to be followed by bismuth and astringents, with opiates to control the pain—a very unsatisfactory manner of management in most cases when compared with the treatment I have already suggested. When there is great relaxation, and the discharges persist in spite of the treatment advised and an astringent seems desirable, a decoction of erigeron canadense may be employed for such purpose, as it does not irritate the intestinal mucous membrane. When the plant cannot be obtained, two- or three-drop doses of the specific medicine diluted in water may be tried.

The *general* management of enteritis is an important subject. The patient should not be permitted to rise to stool, however urgently he may request it, as every time the erect position is assumed the intestinal irritation is increased and the frequency of the evacuations encouraged. A bed-pan should be employed to receive the evacuations, and the patient should be enjoined to avoid defecation as long as possible in order to encourage rest and quiet for the bowels.

The *diet* should consist of mutton broth, pancreatinized milk or milk and lime-water (two or three ounces of lime-water to the pint of milk). After the disease has been arrested the yolk of eggs which have been boiled an hour or partially cooked (soft-boiled) eggs, raw oysters, scraped beef with toast and well boiled rice may be given, care being observed, if the case has been a severe one and the diarrhœa has continued until the patient has become prostrated, to return to a solid diet cautiously.

Care should be observed to prohibit an early return to active habits for fear of provoking a relapse. During the active stage of the disease the patient should refrain from drinking much water, as this is apt to increase the number and frequency of the evacuations. Thirst may be appeased by allowing small portions of ice held in the mouth, or by minute doses of rhus tox.

In malarious districts the practitioner should be on the alert for malarial complications, properly selected remedies often failing under such circumstances to produce their specific effects. It is always a safe plan, when there is the least suspicion of malaria, to administer a two- or three-grain dose of *arseniate of quinia* 3x three times daily, in alternation with other remedies.

Chronic Intestinal Catarrh.

Synonyms.—Chronic Catarrhal Enteritis; Chronic Diarrhœa; Chronic Catarrhal Entero-colitis.

Etiology—Chronic enteritis is due in the majority of cases to

long-continued use of improper food under unhygienic circumstances. Old soldiers, who have been compelled to subsist during arduous campaigns upon hard-tack, salt-pork, underdone beans and such diet while sleeping on the ground and exposed to wet and cold, are the ones most subject to the disease. Repeated acute attacks badly treated may be followed finally by chronic diarrhœa; and certain cachetic conditions, such as gout, may predispose an individual to it. It often arises as a complication of chronic disturbance of the portal circulation, such as that arising from hepatic cirrhosis or splenic hypertrophy. Cancerous or tubercular disease of the intestines may be attended by it.

Pathology.—The small intestine and, usually, a portion of the large bowel are involved in the morbid change. The entire wall of the intestine is frequently thickened by hypertrophy of its coats, and the mucous membrane is reddened and congested and covered with a layer of tenacious mucus. The glandular elements are also hypertrophied and stand out in relief, ulceration of the follicles occasionally occurring. Sometimes atrophy of the intestinal canal is present instead of hypertrophy, and the mucous membrane is leaden-gray in color and the glandular elements shrunken. The villi may be pigmented in patches, imparting a "shaven beard" appearance to the mucous membrane. Where ulceration of the follicles is general the intestine may be perforated with a honey-comb suppurating surface, and perforation or hemorrhage may be the result. Adhesions of the peritoneal surfaces of the intestines may occur and serous exudation may give rise to ascitic accumulation in the peritoneal sac.

Symptoms.—The symptoms resemble those of acute enteritis, though there may be periods of constipation, during which there is more than usually severe distress. Attacks of diarrhœa follow indiscretions in diet in mild cases and food of any kind provokes distress, gastric and intestinal, when the condition is aggravated. The stools are thin and gruel-like, containing mucus and débris of undigested food, with streaks of blood and pus whenever there is extensive ulceration. The evacuations are preceded and accompanied by severe griping pain in the abdomen, and painful tenesmus attends when the irritation extends low in the bowel, it nearly always appearing two or three hours after eating. There may be from one to eight evacuations during the day, and during aggravations, which may arise from cold or dietary indiscretion, there may be a much larger number. Flatulence and borborygmus are common to such cases and hypochondriasis and melancholia are frequently present. Gradual failing of health and strength result and during advanced stages the patient may become extremely emaciated and prostrated. Ascites may then arise, especially where there is hepatic complica-

tion; and later anasarca may occur. The skin becomes sallow and pallid, the pulse feeble and rapid, and the patient may finally die with typhoid symptoms.

Prognosis.—There is little hope in aggravated cases, though recovery may follow treatment begun at an early period. Perforation of the bowel is an occasional accident.

Treatment.—Attention to *diet* is one of the most important matters to be considered in treatment. Food should be liquid in form, and skimmed milk is probably most appropriate when diluted with lime-water. Sometimes the milk is more acceptable when boiled. An excellent plan is to put the patient upon an exclusive milk diet for several weeks until curative treatment is under way. Beef peptonoids, scraped raw beef, beef meal and similar articles are useful. Fatty and saccharine kinds of food should be avoided and only a small amount of farinaceous food should be allowed. In the treatment of such cases the practitioner should consult standard authorities on dietetics for a variety of foods. Fats, sugar, very rich milk, green vegetables, acid fruits and dried fruits, nuts, shell-fish, pork, veal, coarse bread, pastries, cakes and desserts of every description should be discarded.

This is an excellent place for the administration of the *dry diet*. From some limited experience I am convinced that severe cases may be thus controlled, provided they be not too far advanced. If the patient can be induced to persevere in this for a few weeks little medicine may be needed to perfect a cure. The great difficulty is to imbue the subject with enough faith to hold out to the end. The diet should consist of stale bread, thoroughly masticated, with two ounces of plain claret four or five times a day, and *nothing else*. All fluids should be sedulously avoided as well as solids.

To remove accumulations of tenacious mucus, *hydrozone* may be of service, a drachm being diluted with four ounces of distilled or boiled water, two ounces of the amount being taken before breakfast for two or three consecutive mornings, the agent to be repeated as circumstances seem to demand. *Glycozone* may sometimes be substituted with advantage.

To encourage reparation of the diseased mucous membrane, *berberis aquifolium* is an excellent remedy, especially if there be ulceration to contend with. *Epilobium* continued for a long time often affords satisfactory results in cases not too far advanced. *Kaki* is another remedy of especial service for this purpose, especially when a recent preparation can be obtained. *Rhus aromatica* is also an excellent remedy, especially to relieve acute aggravations. *Mercurius* dulcis or corrosivus 6x may be relied upon to alleviate irritation of the colon and heal abraded surfaces there. Severe colicky

pains may call for *colocynth*, especially when the neurosis is about the umbilicus, and *collinsonia* if the rectal or hypochondriac region is the seat of pain. Reference to Dynamical Therapeutics will afford more information as to details in the use of these remedies.

The tissue remedies sometimes exert an excellent influence in these cases. Where there is emaciation and anæmia *calcium phos.* 3x may prove an excellent remedy. *Calcium sulphate* 3x may be applicable to cases where there is profuse purulency, and *natrum chlor.* 3x where there is much watery discharge.

Massage over the abdominal region, where there is not too much tenderness, and general massage, as well as local and general faradization, are useful to assist in a cure. The skin should be protected with flannel and sudden changes should be avoided. A winter residence in a warm climate is always appropriate.

Phlegmonous Enteritis.

This is a very grave disease and one that usually terminates fatally. The abdomen is distended, tympanitic and very tender to touch, there are pain and tormina in an intense degree, the patient breaking out into profuse perspiration, with frequent and violent vomiting, the ejected material becoming fecal in most cases late in the disease. The pulse is small, rapid and compressible and the temperature elevated (103° to 105° F.). There is marked and rapidly progressive prostration in fatal cases, the countenance becoming shriveled and the extremities cold, hiccough and jactitation coming on later, and finally collapse and death. In favorable cases diarrhœa sets in, the tongue becoming dry, red and glazed and the patient greatly prostrated for a time, the symptoms afterward gradually ameliorating and slow recovery following.

Treatment.—*Potassium chloride* 3x, *echinacea* and *baptisia* are the most rational remedies. Solid food should be avoided and the patient should maintain the horizontal position until convalescence.

Pseudo-Membranous Enteritis.

Synonyms.—Diphtheritic Enteritis; Croupous Enteritis.

Definition.—An intense inflammation of the intestine, characterized by an exudate and destructive processes involving the mucous and submucous structures.

Etiology.—Many causes may result in this form of intestinal inflammation. It is not an unfrequent attendant of the infectious fevers, especially of typhoid fever, scarlatina, pyæmia, etc. It may attend the last stages of such chronic affections as cirrhosis of the

liver, cancer, Bright's disease and other cachetic conditions, and may be present in poisoning from various mineral agents, such as mercury, arsenic and lead.

Pathology.—The exudate is thrown out upon the mucous surface, involving it in in a state of coagulation necrosis. Sometimes it is extensive and crust-like and at other times it may constitute a thin film, the mucous membrane being necrotic in both instances. In other cases the exudation appears in small amount about the openings of the solitary follicles, small ulcers corresponding to these openings being scattered about over the mucous surface. Sometimes the follicles are capped with a raised diphtheritic membrane.

Symptoms.—The symptoms vary greatly in character and severity. Sometimes, as when the exudation occurs in the terminal stages of infectious fevers and other constitutional conditions, there may not be much inconvenience from it, while in toxic cases intense pain, with diarrhœa, may attend. Sometimes the presence of the disease will not be suspected until accidentally observed during autopsy. Shreds of membrane may be voided in the fæces.

Treatment.—*Echinacea* and *lachesis* fortify the system against necrosis, and *potassium chloride* against plastic exudation. *Colocynth* alleviates intestinal pain and tenesmus. A liquid diet provides against danger of perforation and destructive mechanical action, and rest in the recumbent posture favors restoration by providing against undue peristalsis.

Mucous Colitis.

Synonyms.—Membranous Enteritis; Tubular Diarrhœa; Mucous Colic.

Definition.—A chronic disease of the colon of mild character, characterized by the formation of masses of tenacious mucus, which may be voided in long, stringy, irregular masses or in the form of tubular membranes.

Etiology.—Some derangement of the mucous glands of the colon is the cause of the peculiar secretion. What the exact pathological condition is remains a question, rectal pockets and papillæ often being attended by it. It is most common in women of nervous, excitable temperament, hysterical women or neurasthenic men being favorable subjects, though it may occur occasionally in children.

Pathology.—There are few if any pathological changes to be bserved, though the lower inch of the rectal mucosa may be con-

gested. The masses of mucus may often be seen through the speculum, sometimes deposited in irregular layers, sometimes in flakes and sometimes in tubular form.

Symptoms.—Paroxysms, characterized by abdominal pain and rectal tenesmus, come on at intervals of a month or more, sometimes lasting for several days, during which the mucus is voided. The subjects are usually hysterical or neurasthenic and they are frequently hypochondriacal. There is no fever.

Prognosis.—The disease runs a chronic course and may continue for years without serious results to the general health, though the patient may be in a condition of semi-invalidism through the attending neurasthenia.. Much benefit, and often a cure, may result from rational treatment.

Treatment.—As rectal irritation is liable to be the chief provoking cause, careful search must be made for concealed rectal pockets, papillæ and ulcers. When such pathological conditions are found they must be radically treated, the pockets and papillæ being excised and the ulcers healed. This treatment should be followed by daily copious flushing of the lower bowel with a saturated solution of sodium chloride in water, the water to be lukewarm, especially in female subjects, the daily flushing being continued for a year. *Electrolysis* of the lower bowel through the salt-water injection is an excellent curative measure. It should be applied twice weekly. Internally, the following prescription may be used steadily for months: ℞ Specific sambucus canadensis ʒii, specific phytolacca ʒi, fluid extract berberis aquifolium ʒi, fluid extract grindelia squarrosa ʒi, aqua ad ℥vi. S. Take a teaspoonful before each meal and at bedtime—four times daily.

Ulcerative Enteritis.

Ulceration of the intestines may arise from numerous causes. Sometimes the symptoms are prominent and sometimes hardly detectable, the disease running a latent course and the condition finally being accidentally observed during autopsy, this being the first revelation of the existence of a lesion. The following kinds of intestinal ulceration have been noted:

Peptic or round ulcer of the duodenum occurs in about one in forty of the cases observed, the majority affecting the stomach. There is less vomiting here than in peptic ulcer of the stomach, and the pain which follows eating comes on later than in the gastric variety and is not so severe. There is tenderness over the right hypochondrium and in about one-third of the cases hemorrhage ensues. In other cases there is little pain or discomfort and the disease runs a

latent course, sudden death following perforation of the intestine, the cause of demise being determined by autopsy only. Stenosis of the pylorus with gastrectasis may occur, or peritonitis with or without peritoneal abscess. Sometimes there is obstruction of the common bile duct or pancreatic duct. Perforation is usually announced by several days' severe continuous pain in the right hypochondrium. The *prognosis* is more serious than that of gastric peptic ulcer. The *treatment* should be followed on similar lines.

Duodenal ulceration may follow extensive burns of the skin. This depends upon some peculiar sympathy which is not well understood. The intestinal complication comes on in one or two weeks after the burn in the form of irregular patches of congestion and ulceration of the duodenal mucous membrane, the ulceration proceeding to destructive action upon the intestinal walls, attended by hemorrhage and perforation with fatal result. The *prognosis* is unfavorable almost invariably.

Embolic ulcers of the small intestine may arise from obstruction of a branch of the superior mesenteric artery, the colon almost always escaping this form of ulceration. Small necrotic areas appear upon the intestinal wall at points corresponding to the terminations of the obstructed vessels, infarction of the mucous membrane and deeper structures producing them. Extensive suppuration of the intestinal wall, followed by peritonitis and perforation, may follow. Intense pain, with profuse, fœtid discharges, typhoid symptoms of rapid rise, collapse and death are the usual symptoms.

Amyloid degeneration of the terminal arteries of the mesentery may be followed by ulcers, which progress steadily to a fatal termination. Permanent obstruction of the arterial supply renders restoration impossible.

Catarrhal and *follicular* ulcers may occur in the alimentary canal, either in acute or chronic form. Catarrhal ulcers are superficial erosions of the mucous membrane of the colon, sometimes spreading widely, resulting, in chronic cases, in induration of the intestinal wall and tending, on recovery, to cicatrization and narrowing of the passage. Follicular ulcers may occur in either the large or small bowel. They consist of single ulcers with excavated edges, but may extend so as to communicate with one another or perforate the bowel. When extensive, they are seldom repaired.

Stercoral ulcers are the result of abrasions caused by irritating fecal material. They are usually found where hardened fecal material is liable to be retained for a long time, as above intestinal strictures, in the cæcum, flexures of the colon, sigmoid flexure or in the rectum. Destruction of the mucous surface with purulent infiltration occurs at points of continued pressure.

Tubercular ulceration of the intestine is of common occurrence in tubercular subjects, secondary infection resulting in pulmonary or genito-urinary tuberculosis. In other cases direct infection may arise from the ingestion of food contaminated with tubercle bacilli, as in the instance of infected milk. Tubercles first develop in the solitary or agminated glands of the ileum and spread from there upward and downward, involving the entire intestinal canal. The mesenteric vessels become involved and the intestine becomes girdled with tubercular deposits, which form along the course of these channels and break down, leaving ulcerated surfaces encircling the intestine at right angles with the longitude of its lumen. The peritoneal surface of the intestine becomes studded with tubercles and the inflammatory exudation furnishes adhesive material to weld the opposing peritoneal surfaces together. Tubercular deposits spread to the mesenteric glands and these become enlarged and nodular. The "girdle" character of the ulcers serves to distinguish them from other forms. Perforation of the bowel sometimes occurs, and healing by cicatrization has been possible.

Syphilitic ulcers occur, in the adult, almost exclusively in the rectum, where they cause progressive fibrous stricture. They are most frequently found in women. Syphilitic ulceration of the small intestine may occur in congenital syphilis. Gummata sometimes form in the intestinal wall, their dissolution being followed by obstinate ulcers.

Uræmic ulcers occur in connection with advanced nephritis, sometimes of gangrenous character, sometimes follicular and again as a result of pseudo-membranous enteritis.

Mercurial ulcers follow poisoning by mercury and are left after the pseudo-membranous enteritis, which then arises.

The ulceration which attends typhoid fever, diphtheria, anthrax and other infectious diseases has already been referred to.

Peritoneal erosion from the pressure of abdominal tumors may result in intestinal ulcers, and a neighboring abscess may perforate the intestine from its external surface and cause ulceration.

Cancerous ulceration of the intestine will be referred to later.

The *symptoms* of intestinal ulceration vary greatly, the location and character of the condition determining its nature and gravity. Limited ulceration of any part of the intestine may be attended by constipation, but diarrhœa is a common symptom when ulceration is extensive. Hemorrhage is a common symptom, though its amount will depend upon the depth of the ulcerative action. Typhoid ulcers and those occurring in the duodenum are attended by the greatest amount of hemorrhage, though perforations of the intestine from without are liable to be followed by extensive hemorrhage. Pus is

always present and its presence is diagnostic. Sometimes it is so limited in quantity as to require microscopical inspection of the fæces to detect it, and at other times it may be present in large quantity, casual inspection affording evidence of it. Profuse evacuations of pus indicate the discharge of an abscess into the intestinal canal. Pus mixed with blood and mucus indicates the presence of an irritable ulcer near the lower outlet, such as that of cancer of the sigmoid flexure or rectum. In tubercular ulceration tubercle bacilli are liable to be found in the evacuations, and shreds of mucous membrane suggest rapid and extensive ulceration, though care must be exercised that portions of undigested food be not mistaken for them.

Pain may or may not be present. Sometimes intestinal ulcers run a latent course and the patient does not experience any pain throughout. Sometimes colicky pains of spasmodic character may attend and are aggravated by taking food. Sometimes the pain is forceful and tenesmic in character, suggesting irritation in the colon or rectum, and is aggravated by motion of the bowels. Sometimes the pain is constant, suggesting peritoneal tenderness. Tenderness may or may not be present. When a small area is sensitive to pressure the symptom is valuable as localizing the seat of ulceration.

Fever and emaciation may be present, depending upon the extent and character of the ulceration. Intestinal obstruction from cicatrization, localized peritonitis, peritoneal abscess and perforative peritonitis are among the *complications* of intestinal ulceration.

Treatment.—A properly selected *diet* stands first among considerations of treatment. A milk diet is best in most cases, though provision should be made against the formation of hard curds by the addition of lime-water. As the upright position encourages peristalsis and thus tends to aggravate intestinal disease, rest in bed is the best course to pursue during treatment. The yolks of eggs boiled for an hour and taken with a little salt may be alternated with the milk diet when this becomes too irksome, and after improvement begins soft-boiled eggs, raw oysters, minced beef and chicken, soda crackers, bread and milk, toast, blanc-mange, custard, junket and wine-jelly may be allowed. Acids, pickles, raw fruits and all indigestible foods should be prohibited, and meals should be limited as to quantity, the patient being fed often and a little at a time—five or six times a day.

When the ulceration is in the upper portion of the alimentary canal, two ounces of diluted *hydrozone*—one or two parts to thirty-two of distilled or sterilized water—should be given two or three times daily to destroy pus microbes, disinfect the alimentary canal

and stimulate reparative action in the ulcers. When the lower bowel is affected, high injections of the same agent may prove serviceable. The constitutional influence of *berberis aquifolium* in ulceration of the soft tissues should be made avail of, and if the ulceration should be syphilitic it may be advantageously combined with *corydalis formosa*. When the small intestines are ulcerated, *kaki* may be of service, and also *epilobium*. *Salicylate* and *subnitrate of bismuth* are to be remembered. Tubercular ulceration should be treated with *bovinine*, such auxiliary treatment as special cases demand being added. Ulceration of the colon and rectum may demand the use of the salt-water electrode with galvanism applied every second or third day for months. Colicky pains may demand *colocynth*, *dioscorea* or *stannum* 3x. In follicular ulceration of the small intestine *kali bichrom.* 3x may be of service, and *mercurius cor.* 6x exerts a similar influence upon the colon. The pain of cancerous ulceration demands *echinacea*.

The *galvanic rectal douche* is a simple arrangement for conveying electricity directly to the interior of the bowel. In ulceration of this part, and in pus pockets communicating with the lower bowel, it is a superior means of treatment. However, it is efficacious in confirmed torpidity of the lower bowel, proving curative in long-

standing constipation as well as in chronic catarrhal conditions of the rectum and colon.

In addition to an ordinary galvanic battery with the usual sponge electrode, the apparatus consists of two yards of half-inch rubber

hose, three yards of copper wire (the size of an ordinary knitting needle) and a small glass, rubber or tin funnel (glass or hard rubber being preferable to the metal on account of non-conducting properties).

In using the patient is instructed to lie, either on a cot or on a rug on the floor, in the Sims position, the floor being better. One end of the rubber hose is oiled and inserted into the rectum an inch or two and the funnel is adjusted to the other end to facilitate the introduction of the water when all is ready. The copper wire is now passed through the funnel and into the tube and carried along until the lower end reaches the lower extremity of the tube, though it should not protrude from it, as a painful effect may result from the current when the metal is in contact with the mucous membrane of the bowel. The upper end of the wire, which should be a yard or more longer than the tube, should be attached to one pole of the battery to be used, the selection to be made according to the condition to be treated. A conducting cord should now be fastened to a moistened sponge electrode and attached to the opposite pole, when all is ready for operation. After the tube has been introduced a strong solution of warm salt water should be poured into the elevated funnel, the patient meanwhile holding the sponge upon the naked abdomen and stroking it backward and forward over the entire abdominal surface. As long as the water flows into the tube freely it should be gradually added, a gallon sometimes being received without much discomfort. The current may be ten or twelve milliamperes in strength—all that a common portable 32-cell McIntosh battery can generate, and the seance can be continued for twenty minutes or half an hour. Powerful tenesmus may attend, but the patient should be instructed to resist this and retain the fluid as long as possible in order to derive the full benefit of the treatment. Its value in many morbid states of the lower bowel and pelvis is not half appreciated.

Dysentery.

Synonyms—Bloody Flux; Recto-Colitis.

Treatment.—A specific and non-specific inflammation of the large intestine, attended by fever, tormina and tenesmus, and characterized by frequent evacuations of tenacious mucus mixed with more or less blood.

Etiology.—Dysentery is divided into non-specific or that form for which a specific cause has not been determined, and specific, tropical or amœbic dysentery, due to the presence of the *amœba coli* in the bowel. The drinking of stagnant water is believed to

originate the disease, and epidemics arising during the late summer and early autumn, when the dry season is prevailing, may be due to low, stagnant water from which the drinking supplies are obtained. This is especially liable to be the case, the dysentery being of severe form, when many are congregated together, as in military camps, the disease probably being spread by contamination of the drinking water from neighboring cesspools. Doubtless the same causes prevail in a more restricted degree in rural districts, where sanitary provisions do not insure immunity from contamination of this kind. Dr. W. C. Cooper *(Eclectic Medical Gleaner)* has recently reported that the precaution of boiling the drinking water in an epidemic of the disease where he practiced (in 1896) did not prevent it from spreading to those who confined themselves to the use of that water after the epidemic began; but it is to be recollected that the foundation may have been laid for weeks before the disease manifested itself—that those who suffered from attacks might have all been contaminated before the first case became known. The germs of the disease seem to sometimes exist in fruits and vegetables, especially unripe fruits, or there must be a variety which may arise from fermentation and local irritation, outside of any specific microbic cause, from eating unripe fruits and such vegetables as cucumbers, as attacks often follow so soon upon the eating of such articles as to render it obvious that their use has direct connection with the malady. After an attack of dysentery one is apt to suffer regularly at the same season year after year, unless successfully treated early for one or two seasons. Sudden arrest of the cutaneous secretion may precipitate an attack of dysentery, especially during the heated term. Antihygienic surroundings other than those already mentioned, such as foul air, depressing agencies, malarial influences and acute febrile attacks, seem to predispose to it.

Pathology.—In *follicular dysentery* the membrane of the lower colon and rectum becomes congested, swollen and reddened, the redness varying in intensity in different portions of its surface from a bright-red color to a dusky or purplish hue; the entire surface is covered with tenacious mucus, and the follicles are enlarged from serous infiltration and proliferation of the new epithelial elements. As the inflammation progresses destructive action follows, and necrosis may begin in the follicles and small ulcers form. As these spread several may coalesce to form irregular ragged ulcers, and in severe cases these may undermine the mucous membrane and penetrate the submucous and muscular coats. Complete perforation of the peritoneal coat occurs in rare instances. In epidemics of typhoid dysentery there is apt to be marked necrotic tendency about the affected tissues, and the destructive action is much more extensive than in spo-

radic cases. The pathological changes in amœbic dysentery are also liable to be of serious character.

Diphtheritic dysentery sometimes occurs, when the ulcers arise independently from the follicles and are more extensive and destructive. The ulcers are covered with a yellowish, fibrinous exudation, the longitudinal axis corresponding to the fold of mucous membrane between the pouches, and the first layer is penetrated and the submucous coat infiltrated with pus. Serious undermining of the membrane is apt to occur in this form, it being more destructive than follicular dysentery. Sometimes tubular casts of a considerable portion of the colon may be formed by the pseudo-membrane.

In *amœbic dysentery* the amœbi are found in the bottoms of the ulcers and in the neighboring bloodvessels and lymph-channels. Purulency is not marked in this form, and destructive local action is not so liable to prove serious as secondary abscesses, which develop in the liver, apparently from the transmission of the amœba coli through the portal capillaries. Abscess of the liver occurs in about one-fifth of all cases of tropical dysentery.

Symptoms.—The location of the inflammatory action determines, considerably, the character of the symptoms. In a large majority of all the cases the lower part of the colon and the rectum are principally involved, and the symptoms are more local than constitutional, while, if the upper colon is exclusively involved, as is sometimes the case, there are few local symptoms, while such constitutional manifestations as chill and febrile action are marked.

In that form which affects the lower portion of the large gut tormina and tenesmus are frequent and urgent symptoms, the periods of tenesmus being attended by the evacuation of a tenacious, glairy mucus resembling the white of an egg in appearance and consistency, and being more or less tinged with blood. The blood varies in quantity, sometimes being mere streaks of pink color in the clear mucus, and in other cases constituting the principal portion of the stool. The tenesmus is sometimes so severe as to cause extreme suffering, the patient remaining constantly on the stool or bed-pan, straining, as though there were a foreign body in the rectum to be evacuated, and returning almost immediately after leaving it, in response to the intense dragging sensation in the rectum. The tormina or griping is usually correspondingly severe, the pain ranging along the course of the colon, but being most common along the transverse colon just above the umbilicus. The rectum is usually the seat of severe burning pain, and the rectal mucous membrane is sensitive to the touch of the finger, a rectal speculum or the tube of a syringe. Sometimes, especially in children, there is complete prolapse of the rectum during the tenesmus, and the anus in most cases

presents a purplish, swollen, ecchymotic appearance. The bladder and urethra sympathize, and the rectal evacuation may be accompanied by the passage of a few drops of scalding urine accompanied by vesical tenesmus. Fecal material may appear among the mucus during the first evacuations, and often the disease begins with a feculent diarrhœa; but soon there is constipation, the evacuations being limited to blood and mucus at first, with muco-purulent passages at a later stage of the disease. There may be from ten to two hundred evacuations in twenty-four hours.

There are more or less constitutional symptoms which attend, and sometimes precede, the local disturbance. Sometimes there is loss of appetite, furred tongue, nausea, headache, dry skin and alternating diarrhœa and constipation for several days prior to the development of the disease proper. As the dysentery becomes developed the temperatures rises to 102° or 103° F., and it may rise as high as 105°. If the upper portion of the colon is exclusively involved the disease may be ushered in with a marked chill, though when the region of the rectum is principally involved this is rare. Typhoid symptoms are common in epidemic dysentery, and the patient becomes delirious, the delirium usually being of the muttering, somnolent variety, the evacuations being dark, resembling prune juice in appearance, often containing shreds of broken down mucous membrane, and emitting a cadaverous odor. The tongue becomes brown and dry, the patient dozes with the mouth open, the pulse becomes feeble and rapid, the respiration hurried, the eyes appear sunken, and serious prospects seem to be in store.

Other varieties of *evacuations* than those already described are the large, watery, feculent stools which occur, without pain or tenesmus, when the dysentery is confined to the upper colon; and the masses of mushy, boiled-sage material which are frequently observed, and which consist of semi-digested starch granules which have passed the alimentary canal without complete disintregation. The stools of amœboid dysentery may sometimes consist of bloody mucus, but they are most commonly fluid of a yellowish-gray color, in which are the actively moving amœbæ. There is less tormina and tenesmus than in catarrhal dysentery, and hepatic complication is more common.

Malarial complication is common in malarious districts, the tormina and frequency of the evacuations being regularly better and worse at stated periods of the twenty-four hours, corresponding to the exacerbations and remissions of a malarial fever.

Scrobutic dysentery is a complication of dysenteric inflammation with the ordinary symptoms of scurvy. In this form profuse hemor-

rhages are common, and fatal results are extremely liable to occur within a few days after the onset.

Diagnosis.—There is little danger of confounding dysentery with acute proctitis, as there is scant evacuation in that disease, no elevation of temperature, and little disturbance of digestion. The long-continued intestinal obstruction and general marasmus would distinguish rectal cancer. In diarrhœa complicated with hemorrhoids there might be rectal tenesmus and colicky pains, but there would be an absence of febrile symptoms, and an examination would detect the exciting cause of the tenesmus in the hæmorrhoidal tumors.

Prognosis.—Catarrhal dysentery of non-malignant type is usually a disease of favorable prospects unless bunglingly treated or the patient has advanced far in years. Epidemics of dysentery are sometimes malignant in character, and the prognosis should be guarded, especially in elderly persons.

Diphtheritic dysentery is a much more severe form of the disease than catarrhal dysentery, and fatal results are more common, especially when malignant. Amœbic dysentery always carries with it the suggestion of possible hepatic abscess, with future doubtful contingencies.

Treatment.—The treatment of dysentery will vary, according to the demands of individual cases. Many mild sporadic cases may be aborted by the use of cathartic doses of *podophyllin*. Half a grain may be administered to an adult at a dose, and repeated every three or four hours until its cathartic action is manifested. When this passes off the disease will, in favorable cases, have been arrested. Broken doses of *sulphate of magnesium* answer a similar purpose. In all cases of sporadic dysentery where constipation exists, it is a good plan to precede other treatment with the use of a cathartic.

In epidemics, however, this treatment will fail, and often there will be aggravation from it. We must then rely upon remedies which act more slowly, but which conserve normal processes and tend to restore structure. Opiates and astringents are worse than no treatment at all, as they only temporarily palliate the pain and lessen the discharges, while the opiates finally aggravate the constitutional difficulty by arresting secretion, and the astringents increase the local inflammation. The pain will be controlled better, in the majority of cases, by minute doses of *colocynth* or *dioscorea*, and the local inflammation will be benefited at the same time. A combination of one of these with *aconite* is usually valuable, as the aconite controls the febrile action to a considerable extent and at the same time specifically soothes the intestinal mucous membrane. *Ipecac* is also a valuable remedy here, it being probably the most generally

applicable agent we possess in intestinal irritation of this character. As a general proposition then we will prescribe in ordinary cases the following combination: ℞ Specific aconite gtt. vii–x, specific colocynth gtt. i–ii, specific ipecac gtt. x–xv, water ʒiv. M., and order a teaspoonful every hour. In all cases the patient must be enjoined to maintain the recumbent posture, using the bed-pan and avoiding straining, as aggravation is almost certain to follow unless these injunctions are observed.

Sometimes *colocynth* will answer alone, especially if the tormina is the leading feature of the case and there is much blood evacuated. Sometimes the tongue is pointed and reddened at the tip and there is constant nausea and restlessness. Here we will expect good results from a combination of aconite and *rhus tox.*, especially when the nervous disturbance amounts to delirium. Twenty drops of specific rhus tox to ten of aconite in four ounces of water, teaspoonful at a dose, will be appropriate proportions. Typhoid dysentery may demand the use of *echinacea*, especially where there is evidence of extensive destruction of the intestinal mucous membrane as suggested by prune-juice discharges or shreddy material in the stools with cadaveric odor. The dirty pasty coating on the tongue may demand *sulphite of sodium*, the brown coating may call for *sulphurous acid*, and the beefy tongue may call for *muriatic acid*. *Baptisia* may be substituted advantageously for echinacea at times probably, the specific indications recognized for it by many being the prune-juice character of the intestinal discharges. Excessive hemorrhage may suggest the need of *rhus aromatica*, and persistent ulceration, as indicated by the presence of pus in the stools, may require the use of attenuations of *mercurius corrosivus*. The rectal injection of laudanum constitutes an old-fashioned measure which had better be avoided, as narcosis and other unpleasant results follow, while no curative action can be expected from it.

The use of *hydrozone*, both as enemata and internally, is highly recommended in dysentery. A drachm of Marchand's hydrozone may be added to four ounces of sterilized water and two doses made of it, one being taken in the morning and the other in the evening. Where it seems necessary to disinfect the lower bowel it may be used as an enema, the patient lying on the right side to facilitate its passage into the intestine. High injections of large quantities of the same strength may be used for similar purposes. It destroys accumulated mucus and disease-germs, thus arresting suppuration and fermentation, and does not interfere with other means.

Periodicity in dysentery should be recognized, and proper antiperiodic treatment be combined with the measures recommended for the control of the dysenteric symptoms.

The *diet* should be carefully regulated. Horlick's malted milk will supply every purpose when it can be taken without objection from the patient. Scraped raw beef is preferred by some and may be allowed once a day where desired, and plain milk, boiled and diluted with lime-water to prevent hard curds is excellent. Some allow the albumen of raw egg, beaten in sherry wine. Starchy food should not be given, as the digestion of starch seems to be interfered with during the disease. The quantity of food allowed should be very small at one time, and no cold food should be taken, the drinks also consisting of warm mint tea and other bland decoctions or infusions administered warm. Return to solid food should be gradual, and fruits should be taken with caution for a time and only after having been cooked.

Cholera Morbus.

Synonyms.—Cholera Nostras; Sporadic Cholera; English Cholera.

Definition.—An emeto-catharsis, characterized by simultaneous vomiting and purging of watery material, with intense thirst, pain in the abdomen and legs, coldness in the extremities and prostration.

Etiology.—Cholera morbus is a disease of hot weather, and is usually due to some disturbance of the digestive processes from the eating of unripe or over-ripe fruits or vegetables in season at such times, though indigestible food may excite it in susceptible persons at any time of the year. Sudden checking of perspiration by exposure to draughts while heated, or by drinking iced liquids, may bring it on, and contaminated drinking water doubtless causes it, hot, dry weather being the time when the source of water supply is most apt to suffer from stagnation. Sometimes the disease seems to assume epidemic proportions, though it is usually sporadic. Males are more frequently affected than females, and extreme old age provides considerable exemption.

Pathology.—This varies considerably, there sometimes being no ocular evidence of structural change or vascular disturbance, while again evidence of acute enteritis may be present. Cerebral anæmia is sometimes found upon autopsy, and effusion into the ventricles may attend. Few opportunities of autopsy have been afforded, as the disease is seldom fatal, and little study therefore has been made of post-mortem appearances. As the disease is of short duration, what changes take place are liable to be largely vascular.

Symptoms.—The attack usually comes on at night, and is announced by vomiting and purging, this sometimes being preceded by

several hours of premonitory symptoms, such as nausea, headache and vague abdominal distress. The vomiting is violent and projectile, and comes on suddenly and without premonition, and the retching is often accompanied by simultaneous gushes of serous diarrhœa of acid or bilious character. Though the patient is thirsty and drinks greedily the fluid is immediately rejected, and solid food is also vomited as soon as swallowed. Gushing evacuations from the bowels follow one another in quick succession, the evacuations being watery and of a mouse-like odor. The evacuations are sometimes large and light colored, and at others small and dark in appearance. Abdominal pain accompanies and precedes the evacuations and vomiting, and, in severe cases, cramps in the lower extremities, especially in the calves of the legs and feet, attend. The urinary discharge becomes arrested, this probably being due to excessive drainage through the bowels, though albumin and desquamated epithelium may be found in the renal secretion. Prostration may become marked, and it may seem as though a speedily fatal termination might be imminent, but after a few hours the symptoms suddenly and spontaneously abate and the patient recovers without interruption, being apparently as well as usual in a day or two. In some cases, however, the pulse becomes feeble and flickering, the respiration and voice feeble and the surface icy cold, and the patient passes into a stage of collapse and dies, the mind being clear throughout. Febrile symptoms are absent, except when pyrexial action attends a short period of convalesence, this sometimes occurring, the symptoms being mildly typhoid in character, the condition being termed the *reactionary fever*. The stools usually become normal within a day after the attack.

Diagnosis.—There is little danger of confounding this disease with anything except Asiatic cholera, and this would occur only when Asiatic cholera was prevailing as an epidemic. In Asiatic cholera it is to be remembered that there is no fecal odor to the stools, which are rice-water in appearance from the commencement, while in cholera morbus the stools are at first fecal. Irritative poisoning may present us with symptoms similar to those of cholera morbus, but the history of the case will probably afford light on the subject; the mouth and pharynx are liable to be hyperæmic, and the evacuations will contain more or less blood, a condition never found in cholera morbus. Analysis of the vomited material may decide the question, through detection of the presence of corrosive drugs.

Prognosis.—Cholera morbus is seldom fatal. Though a severe disease it is likely to terminate spontaneously is a few hours; though treatment will usually arrest it promptly and prevent a very unpleas-

ant experience for the patient. Elderly persons are the ones in whom there is the most danger of serious results, and if collapse or the algid state should be developed the danger is imminent. The prevalence of an epidemic of intestinal disease coincident with an attack increases its gravity.

Treatment.—The treatment of cholera morbus is simple and effective. As soon as the stomach and bowels are ridden of irritating food and fecal material, and even before, the following prescription may be administered in teaspoonful doses every fifteen minutes, until the vomiting and purging cease: ℞ Specific rhus tox. gtt. xv, Lloyd's aconite gtt. x, sterilized water ʒiv. Mix.

The following prescription may be useful to destroy fermentative products and cleanse the alimentary canal of irritating material: ℞ Marchand's hydrozone ʒi, sterilized water ʒii. Mix, and administer at a single dose.

Heating and stimulating applications to the extremities may be required when there is great coldness of the surface with other symptoms of collapse, and in malarial districts recovery from the active symptoms should be followed by the administration of fifteen-drop doses of the green-plant tincture of *grindelia squarrosa*, repeated three or four times daily and continued for a week or more.

Cancer of the Intestine.

Primary cancer of the intestine occurs in from four to eight per cent of all cases of cancer, the colon being most frequently involved, the rectum being the next most common seat of location, then the anus, cæcum and sigmoid flexure. The duodenum and jejunum, finally, are involved, coming last in order of frequency.

Etiology.—Intestinal cancer seldom arises before middle age, though it occasionally occurs before thirty. Its usual subjects are between forty and sixty, both sexes being liable to it, though it is stated that rectal cancer is more common among males than females. It is impossible to ascribe a specific etiological factor, long-continued irritation probably predisposing to it. Thus, a person who has long been the subject of rectal ulceration, hemorrhoids, pockets or papillæ is more liable to finally develop rectal cancer than one who has been previously without irritation there. The sigmoid flexure and cæcum, on account of peculiar location and shape, are certainly more subject to irritation from fecal movement than some other parts of the intestinal canal. Without doubt the pernicious habit of administering cholagogues, which has been in vogue so long, has something to do, occasionally, at least, with

the occurrence of cancer of the upper portion of the intestinal canal.

Pathology.—The growth commonly arises in the mucous membrane and develops in the submucous tissues, the infiltration extending around the intestine and forming a band of constriction, which narrows, more or less, the calibre of the gut. The constricting band may vary from an inch to three or four inches in width, the intestinal wall becoming infiltrated and indurated about the narrowed portion. In some cases there may be projections of ragged masses into the bowel, especially about the rectum, and fungous growths may project from the anus. Prior to ulceration, scirrhous cancer presents a firm, smooth, nodulated appearance; encephaloid, a soft, vascular aspect, without much tendency to ulcerate or obstruct the bowel. When ulceration occurs, scirrhous is marked by smooth ulcerations with hard, deep edges, and encephaloid by fungoid masses which spring up over the ulcerated surface, interspersed with nodulated and lobulated tumors. In many cases scirrhous and encephaloid may be mingled in the same growth. Secondary cancer is apt to arise in neighboring organs, and the rapid growth of these may obscure the symptoms of the primary tumor. The liver is a favorite location for secondary carcinomatous development, and when this occurs the intestinal disease may come to nearly a standstill, the concentration of pathological energy seeming to be transposed to the seat of secondary growth. Stricture of the intestine is a common condition in scirrhus, and distension of the bowel above this point from impacted feces usually attends. Catarrh of the intestinal mucous membrane in the neighborhood of the cancer is common. Rapid infiltration of the mesentery and neighboring organs may bind the affected part to surrounding tissues, dragging and confining various parts in a firm mat; perforation may ensue from rapid ulceration; fistulæ are sometimes formed between the neighboring viscera; hemorrhage may arise from destruction of branches of intestinal arteries, and various other sequelæ and complications may arise from the extension of the cancerous growth to new localities.

Rectal Cancer.

Symptoms.—The symptoms may best be described by dealing with various portions of the intestine separately.

Duodenal and *jejunal* cancer are marked by symptoms similar to those of cancer in the pylorus. There is often vomiting, though it comes on several hours after eating. The ejected material is coffee-ground in appearance, there is a movable, pulsating tumor in the epigastrium resembling that of pyloric cancer to the touch, and

there is cancerous cachexia with or without marked icteric symptoms, due to obstruction in the biliary duct. Hemorrhages may occur and hæmatemesis or melæna follow, though the bleeding is rarely profuse.

Cancer of the *cœcum* is signalized by pain in the region of the cæcum, with the presence of a prominent tumor, consisting of accumulated feces and cancerous growth, the local symptoms being accompanied by debility, waxy color and other constitutional evidences of cancer. Manipulation may assist the retarded feces past the point of obstruction and lessen the size of the tumor, but the cancerous deposit is still perceptible on palpation. The obstruction is progressive, and permanent impaction of feces finally ensues. Tympanitic dullness is elicited by percussion over the tumor.

Rectal cancer and cancer of the *sigmoid flexure* present symptoms so much in common that differentiation is not easy. Constipation, due to stricture, is usually the first cause of complaint, the bowels moving with difficulty, the fæces being thin and ribbon-like, the evacuations attended by severe burning pain and tenesmus. The pain is often most severe in the sacral region, and from here it radiates along the sciatic nerves into the lower extremities. This is darting in character, and may be so intense as to render existence miserable. In some cases instead of constipation there may be an irregular diarrhœa, signalized by the passage of fæcal material mixed with mucus, pus and blood, the evacuations being attended by severe tenesmus and pain. Fistulæ may be established into the bladder, vagina or urethra, and liquid fæces may escape through these channels. As the sphincter becomes involved it loses its function, and liquid fæces and cancerous products dribble through to excoriate the parts and render the surroundings offensive. On examination the affected part will usually be found to be obstructed by a firm, fibrous band, through which it may be difficult to pass even a small gum-elastic bougie, on account of the rigid and tortuous character of the passage. However, sometimes the rectal walls are relaxed and dilated. In epithelioma of the rectum there may not be much obstruction for a long time after its first development, pain, cachexia and obstruction coming on only at a late period. Obstruction of the colon is indicated by distension of the organ, with hardened fæces, its course being traceable under the fingers through the attenuated abdominal walls. When the sigmoid flexure or upper portion of the colon is the seat of infiltration, the pain may be located in the left iliac fossa and loins much of the time, and when in the rectum in the upper part of the thighs, testes and loins.

In most cases of intestinal cancer cachexia develops early. The patient rapidly loses flesh and strength, there is occasional vomiting,

and constipation is soon marked, though cancer high up in the colon may be attended by diarrhœa. The skin assumes a dirty greenish or waxy pallor and becomes dry, harsh and leathery, remaining wrinkled for several seconds when pinched into rolls; the hair is dry and brittle and the pulse small and feeble. Emaciation progresses swiftly, death from exhaustion being a frequent cause of fatal termination. Profuse hemorrhage is rare in any form of intestinal cancer.

Diagnosis.—The unmistakable symptoms of intestinal cancer are pain, cachexia, constipation and the presence of a tumor within the abdominal cavity; though intestinal cancer may run its course without the detectable tumor. Duodenal cancer is so closely associated with the pylorus and pancreas that confusion may arise as to identity. The character and time of occurrence of vomiting after meals and the fact that the tumor is more movable than that of pyloric or pancreatic tumor will serve some diagnostic purpose. The pancreas is more deeply seated and less movable, and vomiting is not so liable to occur when it is cancerous. A pulsating duodenal cancer may be distinguished from aneurism of the abdominal aorta by the fact that the abdominal aneurism will transmit its disturbance to the femoral artery, while the pulsations of that vessel remain undisturbed in duodenal cancer. In cancer of the small intestine the tumor may be more or less displaced by adhesions and contracting bands, assisted by the weight of the tumor, and this is also true of the transverse colon; but the cæcum, sigmoid flexure and ascending colon are firmly fixed and not so liable to be misplaced. In rectal cancer, after the ulceration has begun, the peculiar fœtid odor of the discharge is important in making a diagnosis.

Prognosis.—The prognosis is invariably unfavorable, the patient succumbing within from eighteen months to four years, depending on the amount of cachexia and intestinal obstruction. Where surgical measures are adopted early in rectal cancer life may sometimes be prolonged.

Treatment.—Where duodenal cancer occasions pyloric obstruction benefit may be derived by the use of *hydrozone* to cleanse the stomach of mucus and other accumulation, and this may be followed by lavage. In any event the use of hydrozone provides for the destruction of purulent accumulation in the intestine and removes accumulated mucus occasioned by the catarahal condition of the neighboring mucous membrane. *Bovinine* supports the strength and lessens the pain, though *echinacea* is the most promising agent we possess for the purposes of alleviating the pain of cancer (ten or fifteen drops of a prime preparation every three or four hours). *Chelidonium* promises much toward a radical cure early; ten drops

of the homeopathic tincture or specific medicine four times daily. Where the rectum is the part affected an operation for the removal of the cancerous mass may be undertaken early when the growth is low, and obstruction, in cases in which this seems inexpedient, may be counteracted through the establishment of an artificial anus by colotomy.

The patient should remain quietly in bed to insure a minimum amount of peristalsis, and the food should be liqu d in form and concentrated in quality.

Intestinal Obstruction.

Definition.—A mechanical impediment to the onward movement of the intestinal contents from compression, twisting, invagination of the bowel, or from the presence of foreign bodies in the passage.

Etiology and Pathology.—Internal strangulation of the intestine is the cause of at least a third of the cases of intestinal obstruction which occur in adults. Adhesive bands connecting portions of the intestine to the abdominal wall may form loops, into which a section of the intestine may enter and finally become strangulated. Meckel's diverticulum may adhere to the abdominal wall to form a loop of this character, and such nooses frequently result from abdominal section, a portion of the bowel remaining adherent to the abdominal wound, or the pedicle of a tumor serving to aid in forming an entangling loop. Other openings, such as the foramen of Winslow, or accidental perforations in the mesentery or omentum, may afford opportunities for strangulation. This accident is most likely to occur to the small intestine. A more frequent cause among children is intussusception or invagination of the intestine, the bowel being telescoped from above downward, so that from an inch to a foot of the gut is incased within the same length below. The ileo-cæcal valve is most commonly telescoped into the colon, though the accident may occur to any portion of the bowel, the condition being due to irregular peristaltic action. As in strangulation the part soon becomes congested and swollen, and peritoneal exudation agglutinates the invaginated part so completely that reduction becomes impossible, the inner section sometimes separating and being discharged per rectum. Necrosis and sloughing of the entire affected part is the most probable termination. This accident is more common among males than females, and more than fifty per cent of the cases are among children.

Invagination.

Volvulus, or twisting of the bowel upon itself, is an occasional

cause of intestinal obstruction. This accident is most liable to occur about the sigmoid flexure, a relaxed state of the mesentery favoring such circumstance, and half a turn being sufficient to cause obstruction. Sometimes two coils of intestine unite to form a knot, which becomes fixed and permanently agglutinated. In old persons shrinking of the mesentery may give rise to twisting of the sigmoid flexure, and resultant obstruction.

Foreign bodies in the intestine sometimes become permanently lodged and so impacted as to cause serious obstruction. False teeth (accidentally, or purposely swallowed—by the insane) may cause fatal obstruction, the ileo-cæcal valve being the point which offers the most resistance. Sometimes buttons, nickels and other coins are swallowed by children and cause blocking of the intestinal passage, though such accidents in children are singularly free from serious results, the foreign material most commonly appearing in the stools after a safe passage through the gut. Large gall-stones sometimes block the passage and give rise to serious obstruction. Some drugs, as bismuth, magnesia and other powders, may combine with fæcal material and form firm masses, which produce obstruction.

Stricture of the bowel from cancerous infiltration has already been referred to. Pressure from tumors, as well as stricture from chronic conditions, is progressive and gradual in character, and the pathological changes are slow in developing and the symptoms are not at first urgent, as in strangulation, intussusception and volvulus.

Symptoms.—Sudden obstruction of the bowel usually occurs while the patient is walking about, and is announced by severe colicky pain, which is localized and intermittent. As the pain continues it becomes more steady in character and increases in severity in its original location, while it is soon more or less diffused throughout the abdomen. Vomiting soon begins and becomes constant and distressing, the vomited material first consisting of the contents of the stomach, then of bile and mucus, and later of stercoraceous material. Obstinate hiccough arises after the vomiting has continued for a time, and the vomiting may finally cease entirely to be replaced by hiccough, which continues until a fatal termination. Stercoraceous material is vomited only when the obstruction arises below the upper third of the ileum. An intestinal evacuation may occur immediately after the obstruction arises, the operation emptying the bowel below the point of obstruction, but constipation afterward attends, all efforts to produce an evacuation proving futile. Tenesmus may arise when the obstruction is low in the alimentary canal, and blood and mucus may be discharged when there is invagination. Tympanites suggests the location of the

obstruction low in the alimentary canal. There is little pain on pressure at first, but later the entire abdominal surface becomes intensely sensitive.

The face presents an anxious, pallid appearance, the surface and extremities are cold, the patient lies on his back with the lower limbs drawn up to avoid strain upon the abdominal muscles and carefully avoids motion for fear of exciting vomiting and abdominal pain. Enteritis of violent character attends volvulus. The mind is clear to the last.

Diagnosis.—In diagnosing intestinal obstruction it is to be recollected that *intussusception* is most liable to occur in children, and when a child who has been previously well is seized with sudden and severe pain followed by vomiting and constipation succeeded by discharges of bloody mucus with tenesmus, the pain and vomiting being urgent and persistent, there are good grounds for suspecting intestinal obstruction of this character. If, in addition to these symptoms, a sausage-shaped tumor appears in the region of the ascending or transverse colon within a day or two, the diagnosis is still more clear. In this form fæcal vomiting is not so common as in some other varieties of intestinal obstruction.

Sudden attacks of similar character in adults with paroxysmal pain at a fixed point, attended by fæcal vomiting and rapidly developing tympanites with constipation, will point decidedly to *internal strangulation*. A history of prior injury, surgical operation or peritonitis, suggesting the presence of adhesions forming entangling loops, will assist in a rational conclusion as to condition. The presence of a tumor is not to be expected here and, though constipation is absolute, there will not be tenesmus or bloody discharges.

Volvulus is more obscure in character, though if the sigmoid flexure is involved it may be suspected by the pain in that vicinity, the marked tenesmus, and mucus and bloody evacuations during the advanced stage.

Obstruction by *foreign bodies* is liable to afford a history of the swallowing of some indigestible, bulky article, and the lodgment is most liable to be made at the ilio-cæcal valve. In fæcal impaction there is a firm, hard tumor in the cæcal region, without vomiting until at a late period, with prior history of constipation. Peritonitis is attended by rise in temperature, while in intestinal obstruction the temperature is not elevated, and is likely to be subnormal. Vomiting is not so liable to attend peritonitis as obstruction, and in peritonitis there is marked abdominal tenderness early. In hepatic colic the pain radiates from the right hypochondriac region, the patient is jaundiced, and there are clay-colored stools with constipation but not obstruction, and the urine contains bile. There is

no fæcal vomiting here and no tympanites. In renal colic the pain radiates from the lumbar region along the ureters to the penis and testes, and there is no interruption to normal intestinal evacuation. A concealed inguinal or femoral hernia may be mistaken for intestinal strangulation, unless a careful examination of the suspected rupture be made.

Prognosis.—This will vary, according to the acuteness of the attack. Chronic obstruction in the adult may exist for many weeks before fatal termination ends the scene, while intussusception of the bowel in a weakly child may cause death within a few hours. Volvulus and internal strangulation are more rapidly fatal than intussusception, and obstruction by large gall-stones and enteroliths is more rapidly fatal than stricture, compression or intussusception. There is possibility that sloughing may occur in intussusception and the lower portion of the invaginated bowel be cast off, with union of the remainder with the regular course of the alimentery canal, with recovery, and there is a possibility that it may be reduced to its normal position if effort be made in the right direction before the parts become agglutinated. On the whole, however, the prognosis is almost invariably unfavorable to recovery.

Such *complications* as enteritis, peritonitis, perforation, ulceration, gangrene, septicæmia, fistula and phlebitis are among the probabilities.

Treatment.—Under no circumstances is attempt to force a fæcal evacuation by catharsis advisable. If there be simply fæcal accumulation the proper use of enemata assisted by the salt-water rectal electrode will more assuredly remove the impaction than the action of cathartics, and its use is permissible when there is actual obstruction. This measure should be tried with both galvanism and faradism, if necessary, the faradic current increasing inverted peristaltic action and thus favoring the relief of volvulus and invagination. A strong decoction of *cimicifuga* root is relaxing and quieting to the intestines, and should be given in wine-glassful doses every hour for a few hours where obstruction is suspected. Inversion of the patient, the body being elevated by the heels to nearly the upright position and maintained there for a time, is highly recommended in intussusception and volvulus, and in this position copious enemata of warm water should be tried. Air, introduced by attaching a rectal tube to a siphon-bottle of carbonated water, may be forced into the bowel, this sometimes serving to relieve an invagination or volvulus, though there is danger of rupturing the gut by incautious application of the measure. After forty-eight hours adhesions are presumed to have taken place, when attempts to remove the fixation will be fruitless. Opiates early are highly recommended, though with doubtful philos-

ophy; but at a late stage they may be administered freely to allay the pain. Abdominal section is justifiable where the diagnosis is confirmed, and it should not be delayed until the strangulation has gone on to gangrene.

Intestinal Hemorrhage.

Synonyms.—Enterorrhagia.

Etiology.—Among the principal causes of intestinal hemorrhage are intestinal ulcers attended by erosion of vessels, and cirrhosis or atrophy of the liver causing obstruction of the portal circulation. Erosion may also be caused by strong drugs, and venous obstruction may arise from pressure by tumors, foreign bodies or hardened fæces. Profuse intestinal hemorrhage may occur from the rupture of an aneurism, and one of the common symptoms of internal hemorrhoids is profuse bleeding from the bowel. The engorgement due to invagination and volvulus is liable to be attended by bloody evacuations, as also are severe inflammations of the intestinal mucous membrane, as in dysentery, enteritis and typhoid fever. Embolism of the mesenteric artery may be a cause of intestinal hemorrhage. A number of constitutional diseases may originate bleeding from the bowels. Among these may be mentioned purpura hemorrhagica, scorbutus, pernicious anæmia, leukæmia, pseudo-leukæmia, septicæmia, jaundice and phosphorus poisoning. The aged may be subject to passive intestinal hemorrhage of obscure nature, men being more liable than women. Melæna neonatorum, or hemorrhage in new-born children, may be due to degeneration of the arteries from syphilitic, fatty or amyloid changes, from puerperal infection and from hæmophilia.

Pathology.—When examined soon after death the intestinal mucous membrane may be hyperæmic or anæmic, depending upon the amount of blood discharged. The intestine usually contains small clots of grumous blood, and when hemorrhage occurs from the surface of ulcers coagula are generally found adhering to them. When the hemorrhage is due to obstruction of the portal circulation there is usually little change from normal in the appearance of the mucous membrane.

Symptoms.—The constitutional symptoms are those of hemorrhage in general. There are sensations of faintness, coldness of the surface, ringing in the ears and syncope, with feeble pulse, pallor, and coma which may end in death. Preceding, attending or following these symptoms, there is an evacuation of blood from the bowels, and this may be attended by pain or other abnormal sensations,

such, for instance, as though warm water was being poured into the abdominal cavity.

The blood varies in color and consistency as it comes from different portions of the intestinal tract. That which issues from the walls of the duodenum is black and tarry in appearance and tenacious in consistency. That from the ileum is usually dark, but it is brighter than that from the duodenum, and returns to its normal color when the clots are dissolved in water. The blood from the large intestine is usually bright-red and fluid. The dark color of the fæces caused by hemorrhage from the duodenum is not to be confounded with the appearance produced by eating huckleberries or taking iron or bismuth. The quantity may vary from a few streaks in the fæces in some cases to an immense quantity—sufficient to cause death in a few minutes, the blood piling up in large heaps between the nates and thighs, in some cases of fatal hemorrhage in typhoid fever.

Diagnosis.—The diagnosis is not difficult, the patient usually finding that he is bleeding from the anus before other attention is called to it, unless it be in inflammatory conditions of the bowel, where the nurse and physician are expecting and dreading it. The location of the hemorrhage may be pretty definitely ascertained by examination of the blood as soon as it is voided, by the general history of the case, and by considering the physical signs referable to the abdomen. When the blood is bright red in color when voided, careful examination of the rectum—under chloroform if necessary—should be made to determine whether or not the seat of hemorrhage is within reach of local treatment.

Profuse hemorrhage during the advanced stage of acute infectious fevers, such as typhoid, yellow and malarial fevers, is an unfavorable symptom, though capillary hemorrhage is far less serious in nature than arterial. General enfeeblement of the constitutional powers is an unfavorable condition for hemorrhage to occur in, fatal results being much more liable to follow than in the robust. A single large hemorrhage may prove fatal, as also may many slight ones.

Treatment.—The treatment will vary, different conditions demanding appropriate measures. Acute inflammatory conditions, attended by destruction of the mucous membrane, will demand a special class of remedies. For instance, the hemorrhage of acute enteritis will be amenable to *ipecac, colocynth, aconite* and *echinacea,* in minute doses frequently repeated, or *rhus aromatica* (specific medicine gtt. x–xx). That of dysentery, when the symptoms are acute, may be benefited by similar treatment. Ulcerative action in the large intestine attended by hemorrhage may be treated with minute

doses of *mercurius dulcis*, though profuse enterorrhaghia may call for more active astringents, such as a decoction of the fresh *erigeron* plant taken freely, or ten-drop doses of the oil on sugar, or *ergot* in appropriate doses. *Tannic* and *gallic* acid, in two- or three-grain doses repeated every hour, assist materially in arresting profuse hemorrhage from the bowels. Sometimes the lesion is in the rectum, where the injection of the bleeding point with a hemostatic (1-4 carbolized oil) through a hypodermic syringe will arrest the hemorrhage at once, when internal remedies may prove of little avail. When there is persistent dribbling from capillary hemorrhoids, they should be systematically treated with interstitial injections of diluted *carbolic* acid (1-4 of olive oil and glycerine, aa.). In urgent cases of rectal hemorrhage enemata of a saturated solution of alum may be retained for their astringent effect until permanent measures succeed. Tamponage may sometimes be resorted to, and scorbutic conditions should be properly met.

Absolute rest in bed is as important as medicine, the recumbent position being strenuously insisted upon—all evacuations being attended to without assuming the upright position—and even turning in bed should be restricted. It may sometimes, in non-inflammatory conditions, be advisable to restrict peristalsis with opiates, though general adherence to this usage is objectionable.

The *diet* should be liquid and nutritious, and it should be administered frequently and in small quantities. Cold applications favor arrest of hemorrhage, and cold drinks and fluid foods are better than warm. In extreme cases ice-bags may be placed upon the abdomen for a limited time.

Typhlitis.

Definition.—Inflammation of the cæcum. The terms *perityphlitis* and *paratyphlitis* are employed to designate, respectively, inflammation of the peritoneal covering of the cæcum (perityphlitis) and inflammation of the connective tissue surrounding it (paratyphlitis). As these conditions are, however, usually complications of appendicitis they are not employed by the best authors as designative of separate diseases. Inflammation of the cæcum may arise as a complication of appendicitis, but the term is here used to designate inflammation of the part, independent from appendical trouble.

Etiology.—Inpaction of fæces is the common cause of typhlitis, the term "typhlitis stercoralis" often being used to designate the condition. Errors in diet are probably the exciting cause, though repeated attacks may be due to colds following an established irritation. The disease is most common among young persons, boys

being more frequently affected than girls. It is always associated with constipation.

Pathology.—The anatomical condition has not been determined, as few if any fatal cases occur.

Symptoms.—Pain in the right iliac fossa, with enlargement, the prominence taking the form of a sausage-shaped tumor, loss of appetite and sometimes nausea and vomiting, are the principal symptoms. There is usually little if any fever, though the temperature may be elevated one or two degrees. The pain and fullness may be accompanied by tenderness on pressure, and there is usually dullness on percussion. The patient lies upon the back, and may flex the right thigh to relieve tension of the abdominal muscles in the affected region. The symptoms are all mild, and gradually subside within three or four days or a week.

Diagnosis.—The diagnosis between this disease and mild cases of catarrhal appendicitis is rather difficult, though the sausage-shaped tumor is regarded by some as a diagnostic feature of typhlitis. Others, however, assert that this symptom occurs as a secondary feature of appendicitis.

Treatment.—Nausea and vomiting may be controlled by minute doses of *aconite* and *rhus tox.*, and *potassium chloride* 3x may be administered as a safeguard against plastic exudation. *Cold applications* may be used over the affected part, though if these are unpleasant they may be dispensed with, warm or hot applications being carefully avoided. Rectal injections of tepid salt water may be employed to assist normal evacuation and, in stubborn cases, the salt-water electrode with galvanism may be used in the lower bowel. The *diet* should be liquid in form and sparing in quantity, and the patient should be kept quiet in bed until a regular condition of the bowels has been established and the pain and fullness have subsided.

Appendicitis.

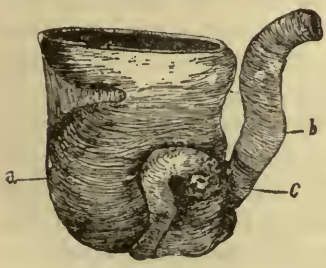
Definition.—Inflammation of the appendix vermiformis.

Etiology.—Appendicitis is most liable to arise in those of early adult life, though it may occur in childhood, even in rare cases during infancy, while it is exceedingly uncommon in advanced age. It has been estimated by some writers that sixty per cent of all cases occur between the ages of sixteen and thirty. Both sexes are liable to it, statisticians differing as to which is most frequently affected. Foreign bodies, such as grape-seeds, orange-pits and other foreign bodies, are probably often causal factors. Some peculiarity of shape or position possibly contributes to the irritation produced by such agents after lodgment there, but where the condition of the part is

normal and there is present no microbic element of disease, the mere presence of foreign bodies cannot be considered an inevitable precursor. Irritation, amounting to abrasion, in such cases, may afford entrance of microbes into the capillaries of the appendix to arouse inflammatory action—in which case foreign bodies would certainly be predisposing causes. Adhesions of the appendix to adjacent viscera might result in teasing tension, which would finally excite inflammatory action. Many cases arise suddenly after the lifting of heavy weights, and such appendicitis is probably thus brought about. Over-eating is liable to be a provoking cause, especially when improper food is taken, this being a frequent cause of the recurrence of the disease after recovery from a first attack. Irritation of the cæcal extremity of the appendix may result in gradual closure of the opening until complete obliteration occurs, a hermetically sealed cavity remaining in the appendix, which may contain elements of fermentation or suppuration.

Pathology.—Two forms of acute appendicitis are recognized, namely, catarrhal and suppurative.

In *catarrhal appendicitis* the mucous membrane is thickened and engorged, and covered with a coating of tenacious mucus, while the cavity contains serum and one or more masses of hardened fecal concretion, and is usually narrowed in its lumen, especially at its cæcal extremity. The entire organ is enlarged, rigid and club-shaped, with its outer extremity expanded, and the peritoneal covering is congested or coated with fibrinous material and adherent to adjacent peritoneal surfaces. When slit longitudinally the mucous membrane rolls outward and the peritoneal covering inward, a position afterward persistently maintained when not interfered with.



APPENDICITIS.
a, cæcum.
b, ileum.
c, appendix.

Suppurative appendicitis is marked by the presence of serum and pus in the walls of the appendix and upon its outer surface. When the suppurative action is not rapidly destructive, the neighboring peritoneum becomes inflamed and covered with adhesive fibrino-purulent material, which binds the folds together in the form of a surrounding wall, and incloses a cavity that becomes distended with pus. Burrowing may now occur and, if the cavity be not drained by proper surgical procedures, purulent material may infiltrate the connective tissue of the mesentery and invade the retroperitoneal tissues, the pus then descending along the psoas or iliac fascia and, appearing externally below Poupart's ligament as an external abscess, burrow

to the peri-nephritic structure, or descend into the pelvis and involve the peri-rectal tissue. In rare cases the pus may penetrate the obturator membrane, pass through the obturator foramen and appear as a gluteal abscess. In most cases the abscess is likely to burst into the peritoneal cavity and cause general peritoneal septicæmia; or, without actual rupture, diffuse suppurative peritonitis may occur. The pus may be profuse or limited in quantity, and when the amount is small it may be circumscribed, and if surrounded by a large amount of inflammatory tissue may remain localized, to undergo absorption or other change. When the amount is very small absorption is possible, though rare.

In other cases there is such rapid and extensive suppuration that sloughing of the appendix occurs, and its necrotic (gangrenous) fragments, along with the contents of the cavity, are discharged into the peritoneal cavity before a limiting abscess-wall is formed, and a virulent peritoneal sepsis is set up, which rapidly ends the scene.

Symptoms. — The symptoms vary, according to the gravity of the pathological condition. Where catarrhal appendicitis is not severe there is constipation, pain and tenderness in the region of the cæcum, nausea and loss of appetite, and where the inflammation is somewhat pronounced there is more or less induration of the abdominal tissues about the cæcal region. Sometimes the symptoms are so slight that the earlier stages pass unnoticed, there being merely slight local pain and tenderness in the right iliac region, In most cases, however, there is an initiatory chill, followed by vomiting and fever. Severe pain usually begins in the right iliac fossa, either steady or of paroxysmal character, and marked tenderness under pressure is found at some localized point in the iliac region, often at McBurney's point, situated on a line with the umbilicus about an inch and a half or two inches from the right anterior spine of the ilium. The temperature varies from 101° to 103° F., and continues elevated for three or four days when, in favorable cases, it slowly declines, the induration gradually passing away, the bowels moving spontaneously and the pain and tenderness disappearing. In severer cases the pain becomes more marked, sharp and diffused, announcing involvement of the peritoneum. The right thigh is drawn up to relieve the abdominal muscles of that side from tension, and in walking the patient bends forward, the erect posture causing pain. The pain may now be radiated, extending over a large portion of the abdomen and involving the bladder, testes, rectum and other viscera. Retention of the urine may occur. The tongue becomes furred, and diarrhœa may set in, especially in children. As unfavorable conditions progress the tongue becomes dry and brown, sordes appear on the lips and teeth, and symptoms of exhaustion supervene. Some-

times, when the appendix turns backward, it is difficult to detect a tumor in the iliac region, a vaginal or rectal examination enabling the practitioner to detect the affected point deep in the abdomen. Where a large amount of pus accumulates within the limiting abscess-wall the abdominal tumefaction may be a marked feature of the case, the abdomen in the region of the cæcum being enormously distended.

The position of the appendix will determine, to considerable extent, the local symptoms and conditions. When it is turned backward, as is often the case, post-peritoneal abscess is very liable to follow suppurative action, and enlargement of the cæcal region is not likely to be noticed. In gangrenous appendicitis the symptoms are abrupt and severe from the start. There is a chill followed by fever, with excruciating abdominal pain marking the rapid spread of peritoneal inflammation, prolonged vomiting of watery fluids, rapid, fluttering pulse, and delirium followed by coma.

Diagnosis.—When persistent pain in the cæcal region attended by elevation of temperature and constipation occurs in patients under thirty years of age, with tumefaction of the part and pain at McBurney's point, there is little danger of confounding appendicitis with any other affection except typhlitis; and here an error of diagnosis would not be serious if radical surgical measures were not attempted too early. The presence of vomiting would add to the probability of appendicitis, and the absence of tumor would not militate against it if other symptoms were marked. When an enlargement in the iliac region with dullness on percussion pointed to purulent accumulation, an aseptic hypodermic needle might be used to decide the question, though due caution as to sepsis and repeated puncture should attend such a procedure. Great haste to decide the question is not necessary, unless the case be one of gangrenous appendicitis, and here it is doubtful whether a diagnosis could be made sufficiently early to afford substantial relief by operation.

Prognosis.—The prognosis of acute catarrhal appendicitis is favorable as to present recovery, though remaining adhesions are liable to perpetuate the difficulty and induce frequent subsequent attacks. Under skillful surgical treatment many cases of suppurative appendicitis otherwise necessarily fatal recover, though the gravity of the disease is not to be underestimated. When the peritonæum is widely involved the chances of recovery are very much lessened, and exhaustive suppuration and final demise are liable to succeed burrowing abscesses when the post-peritoneal structures are invaded. Perforation of the intestine may be followed by recovery, and the pus may find an external opening in front, and the patient recover without surgical aid. However, since the disease has received special

attention from a surgical standpoint and prompt measures for an early evacuation of the pus have been adopted, it has become much less formidable than before.

Treatment.—While appendicitis is a disease in which a knowledge of surgery is an important requirement for its successful management, the physician is also capable of bringing important aid to bear, often rendering surgical aid unnecessary. In recent times, thanks to Schüssler, we possess a remedy which exerts a potent influence against the deposition of fibrinous plastic material, and with it we may be able to avert the pernicious adhesions remaining after ordinary cases of catarrhal appendicitis, as well as even prevent suppuration, if the purulent form be at hand. Limited experience with *potassium chloride* 3x has suggested to me the probability that a large number of surgical cases may be aborted before they become marked, and otherwise portentous states brought to a successful termination by the early and faithful exhibition of this remedy. In all cases it is a perfectly safe one to say the worst of it, and its power to accomplish good is remarkable. In several cases of the kind I have seen the abdominal tumefaction, obstinate constipation, cæcal pain and elevated temperature gradually subside under its influence when, in the opinion of old and experienced surgeons, an operation was urgently demanded. It is perfectly safe to depend upon when there is appreciable (even though slow) improvement of all the symptoms. Three to five grains of potassium chloride 3x should be added to four ounces of water, a teaspoonful of the mixture to be ordered every hour. When this is begun early, and the symptoms continue to increase in severity for two or three days, the probabilities are that the disease is beyond its control, though it can do no harm to continue it until operative procedures are adopted, as no other remedy promises so much, and it must somewhat lessen the amount of destructive action.

In the meantime the patient should remain quietly in bed and the diet should be limited to liquid food, administered sparingly. Cathartics should under no circumstances be allowed, but daily efforts to evacuate the bowels with warm and soothing enemata should be made. Hot applications are to be avoided, and very cold ones are not commendable. Opiates may be allowed in moderate doses, though the patient is better off without them if the pain is bearable. If febrile action is marked properly selected sedatives should be administered in small doses, and where there is prominent suggestion of gangrenous tendency *echinacea* should constitute an important feature of the medication. If the pain and tumefaction increase, in spite of medical measures, surgical aid should be invoked early, and

this should constitute the first resource in gangrenous appendicitis, which may prove fatal in a few hours without.

Proctitis.

Synonym.—Rectitis.

Definition.—A catarrhal inflammation of the rectum, due to local exciting causes, differing from that attending dysentery by the absence of constitutional symptoms.

Etiology.—Sometimes indigestible substances, such as fish bones, particles of skewer, etc., may be accidentally swallowed with the food and pass through the alimentary canal, to become lodged in the rectum, to there excite inflammatory action. This is not an uncommon occurrence when persons who are intoxicated partake of food containing such débris. Several cases of the kind have occurred in my experience, and occasionally an instance has been followed by severe inflammation, resulting in deep-seated abscess in the part. Other foreign substances, such as particles of apple-core, berry-seeds, plum-pits, etc., may also result in such irritation. Hardened fæces, hemorrhoids, sitting long on very cold substances and other exciting causes may be named.

Symptoms.—Tenesmus is the first symptom, and frequently it is the prominent one throughout. Sometimes there are evacuations of bloody mucus attended by straining at stool, burning in the part and shooting pains in the back and loins, or into the lower extremities. A persistent sensation as of a foreign body in the rectum gives rise to repeated efforts at evacuation, and anal prolapsus is very liable to finally result. Hemorrhoids, strangury, headache and other constitutional symptoms and even chronic rectitis may finally attend. Hardened fæces sometimes play an important part in this affection, the rectum becoming impacted with a hard mass, which is too large to pass the anal outlet, and the colon may become filled with stercoraceous material which may be traced along the course of the large intestine by irregular masses felt externally.

Chronic rectitis is attended by the daily discharge of mucus, pus and sanious material, with more or less tenesmus. Erosion and induration of the rectal mucous membrane exists, and the finger detects a hardened, rigid condition upon digital examination. Constipation attends, the fecal material voided being hardened and impacted.

Diagnosis.—In proctitis from local causes there is no fever, while in that which arises in dysentery the thermometer shows an elevation of three or four degrees. The pain of proctitis is also different in character, it usually being confined to the region of the

rectum or radiating to the back or loins, while that of dysentery usually lingers along the course of the colon and is frequently near the umbilicus and paroxysmal, corresponding to the periods of evacuation. An inspection of the rectum will decide whether the symptoms arise from hemorrhoids, and rectal cancer comes on so slowly that it cannot be mistaken for proctitis, while the presence of the characteristic cachexia precedes extreme local irritation.

Prognosis.—The prognosis is good when the disease is properly managed. If the exciting cause be some hard and irritating substance and its removal be neglected, deep-seated abscess, fistula or chronic proctitis may result. Erosions left behind may give rise to chronic rectal catarrh or rectal stricture.

Treatment.—The first important step is to decide whether there be any foreign body present which may be causing the difficulty. If so it should be carefully removed at once, and treatment afterward instituted to control the remaining inflammation. In such cases the finger will be the most reliable and least objectionable exploring agent, and also the best means by which to remove offending substances without injury to the part. Small doses of *aconite* and *rhus tox.*, combined with *collinsonia*, will assist in controlling the local inflammation. ℞ Lloyd's or Worden's aconite gtt. v–vii, green-plant tincture rhus tox. gtt. x–xv, specific collinsonia gtt. x, water ʒiv. M., and order a teaspoonful every hour. Locally, the following may be used as an enema, to be retained until absorbed and repeated every hour or two, according to the urgency of the case: ℞ Echafolta ʒi, water ʒii. Mix. The patient should remain quiet in bed and be allowed only a liquid diet for several days. In chronic proctitis the enema of echafolta should be employed three or four times a day, and collinsonia and echofalta should be administered internally three or four times daily in appropriate doses. *Berberis aquifolium* continued for a long time, in connection with collinsonia or negundium, will be of considerable service as an internal agent.

Periproctitis.

Definition.—Periproctitis is an inflammation—usually suppurative—of the connective tissue surrounding the rectum.

Etiology.—The inflammation may be coëxtensive with that of proctitis or of other diseases which may affect the rectal mucous membrane, such as cancer, ulceration, etc. It occasionally occurs as a result of tubercular infection of the part, or of pyæmic metastasis. Traumatism is its most common cause, the lodgement and neglect of some foreign body in the rectum, or blows near the anus being very liable to result in such a condition.

Pathology.—Suppurative inflammation of the connective tissue occurs at some localized point, and fluctuation may by felt through the rectal wall as the destructive action progresses, the soft part bulging into the rectum. The pus may burrow in the vaginal or vesical wall and establish fistulæ, or a track of suppuration may form completely around the rectum. In other instances the abscess may open into the rectum and a permanent suppurating sinus become established. Proliferation of new connective tissue may result in stricture of the rectum, or proliferating epithelial elements may line the abnormal cavities with mucous membrane similar to that of the rectum.

Symptoms.—Severe pain of throbbing, burning or tensive character, attended by a sense of fullness in the rectum, is the prominent symptom. If the inflammation involve structures near the anus, a reddened prominence, of fluctuating, sensitive character soon develops. Nausea and vomiting may attend severe cases. An examination of the affected part will discover the local signs of abscess, the finger detecting a fluctuating tumor, sensitive to the touch, extending into the rectum. Upon rupture the contents of the abscess are extremely offensive in odor, and they may be mixed with fæcal material.

Treatment.—The most important object is to insure evacuation of the abscess, not into the rectum but through the true skin, near the anus. This demands proper surgical acumen. If treatment is begun early there may be a possibility of avoiding the abscess, especially if it be of traumatic origin and the provoking cause has been removed in the start. For this purpose *potassium chloride* 3x may be administered in the usual manner. The special sedatives, especially *aconite* and *rhus tox.*, are excellent to control serious constitutional symptoms, and if septic conditions arise *baptisia* and *echinacea* should not be forgotten. If chronic purulency, too high for the pus-pockets to be reached from below exist, the persistent use of the salt-water galvanic electrode promises much, if begun at an early date and faithfully used for several months three or four times a week. In such cases the general condition of ths patient should not be neglected, appropriate adjuvant treatment being employed as demanded. *Calcium sulphide, berberis aquifolium* or other antisuppurative may be needed to bring prolonged suppuration to a close.

Hemorrhoids.

Synonym.—Piles.

Definition.—A disease characterized by the formation of vascu-

lar tumors in the lower rectum and about the anus, from varicosities of the hemorrhoidal veins with subsequent inflammatory change.

Etiology.—Straining at stool often causes rupture of one or more of the coats of the hemorrhoidal veins, and this may be followed by permanent hemorrhoidal tumors at the points of greatest dilatation. Riding over rough roads or sitting on cold seats for a long time may cause it by inducing congestion. Pregnancy is often attended by hemorrhoids due to pressure on the pelvic veins, and parturition may be attended by such forcible straining as to result in a permanent hemorrhoidal condition. Constipation is a common cause, both the straining during defecation and fæcal pressure upon the hemorrhoidal veins tending to such result. It is most common in persons beyond middle life, though younger ones are not exempt when exposed to exciting causes. It is more common in single women—those who have not borne children—after the menopause, this period frequently being immediately followed by the appearance of hemorrhoids. Obstruction of the portal circulation from such diseases of the liver as cirrhosis, atrophy or passive hyperæmia, is almost certain to eventuate in hemorrhoids; and influences which cause engorgement of the vena cava, such as cardiac or pulmonary obstruction, are very liable to be followed by it. The abuse of drastic cathartics is often provocative of piles, large doses of colocynth, aloes, etc., frequently bringing on the disease.

Pathology.—Acute hemorrhoids may be nothing more than dilated, inflamed veins, sometimes containing thrombi of coagulated blood. As they continue without proper treatment, however, they may gradually become surrounded by bloodvessels, and the vasa vasorum, from inflammatory action, may become hypertrophied, until the tumors consist of aggregations of dilated bloodvessels with firm fibrous coats, constituting permanent and more or less firm enlargements which, however, increase or diminish in size as their vascularity fluctuates.

Various divisions of hemorrhoids have been made. Those which arise within the sphincter ani and which can be returned to the rectum, to remain there, if prolapsed, are termed *internal hemorrhoids*; while those which arise without the sphincter ani and cannot be carried up, or which immediately return when lifted above the sphincter, are termed *external hemorrhoids*. External hemorrhoids commonly occur just at the verge of the anus, upon the mucous membrane. Sometimes straining at stool is followed by rupture of one of the external hemorrhoidal veins with the formation of a blood-clot within the point of rupture which, if not soon evacuated, is afterward absorbed, the dilated sack afterward becoming shriveled and remaining as a wrinkled tab of muco-cutaneous tissue. Internal

hemorrhoids are sometimes dark blue, livid and non-fluctuating, more rarely bright red and pulsating, suggesting the presence of one or more arterioles, and, when punctured, project a stream of bright red blood in jets. Internal hemorrhoids, then, may be either venous or arterial. Another form of internal hemorrhoids consists of flat mucous surfaces covered by bright red capillary loops *(capillary hemorrhoids)*, which bleed easily and frequently, the amount of blood, though small in quantity in a given time, constituting finally a serious loss to the system. The hemorrhage which occurs in hemorrhoids almost universally proceeds from above the sphincter (internal piles) and usually from capillary hemorrhoids, though the coat of an arterial or venous hemorrhoid may be so attenuated as to give way and allow of considerable loss of blood in a short time, at frequent intervals. Rupture of venous internal hemorrhoids is not rare, and sufferers sometimes rupture them with their fingers, believing that it will afford them temporary relief from pain. The sphincter ani is usually irritable, and when internal hemorrhoids are prolapsed, as often occurs during defecation, spasmodic contraction of the sphincter about them above the protruding masses may cause strangulation, unless the prolapsed tissues are replaced. In other cases, however, the sphincter may be relaxed, and chronic prolapsus of the hemorrhoids may persist.

Symptoms.—Unless strangulation or severe inflammation, with septic absorption occur, hemorrhoids afford only local symptoms. There is a sensation as of some foreign body in the rectum, with pain in different cases, of widely varying character. Sometimes this is dull and aching with dragging sensations about the anus, sometimes it is sharp and piercing as though there were a sharp instrument driven into the anus, and sometimes it is throbbing as though an abscess were forming. In some cases of internal piles the pain is almost confined to the lumbar and sacral regions, and in others it may radiate to the hips and lower extremities. Most cases, if not all, are subject to periods of intense excerbation, due to cold, constipation, riding over rough roads, severe exertion, etc., in which the patient suffers severely, while there are varying periods in which not much discomfort may be experienced. When the disease becomes well advanced and there are extreme changes of structure, the patient becomes a constant sufferer, every period of defecation being one of anguish, prolapse of a mass of distended tumors often attending the exit of the stool and necessitating its return, a performance at which the sufferer after a time becomes an adept. Hemorrhage is common at these times, and, if constipation attend, the suffering is prolonged and intense.

When there is considerable hemorrhage the patient becomes

anæmic after a time, and dyspeptic symptoms are common, probably from reflex irritation. The bladder and urethra commonly sympathise in severe cases, and urination may be difficult and painful. Vesical tenesmus may be almost a constant symptom. In females vaginal pain may be due to such cause. Hemorrhoidal persons are frequently melancholy and morose. Constipation is the rule, though many are subject to diarrhœa.

Diagnosis.—A rectal examination can hardly fail to result in a correct diagnosis. The only rectal tumor liable to be confounded with hemorrhoids is a rectal polypus, which is colorless, prediculated and painless. Venereal growths occur at the edge of the anus but are not painful, and other symptoms of syphilis usually observable will aid in distinguishing this disease from hemorrhoidal tumors, which are smooth, tense and shiny, and usually purplish in color, more or less sensitive to the touch and commonly painful. Hemorrhoids bleed easily, while other rectal growths are not liable to. Capillary hemorrhoids are found within the sphincter and consist of patches of bright red capillary loops, which bleed easily upon being disturbed.

Prognosis.—When hemorrhoids are treated early, before inflammatory changes occur, there is good prospect that specific constitutional treatment may succeed in effecting a cure—at least that the difficulty may be banished for years before it will again appear, unless the patient is continually exposed to its causes. After inflammatory changes have occurred, radical surgical measures are demanded, and medicines by mouth can be palliative only. Capillary hemorrhoids demand radical treatment from the start, and should not be allowed to continue, as stubborn anæmia may arise, to continue for years after the completion of a cure of the local trouble.

Treatment.—Though usually classed as a surgical disease, hemorrhoids may often be successfully treated by the physician with internal remedies. *Collinsonia* specifically influences the tissues of the rectum, and many cases of acute and subacute piles may be permanently relieved by its internal administration. Where constipation attends the following prescription is an excellent one: ℞ Green-plant tincture of collinsonia ʒi–ii, fluid extract cascara ʒi, simple elixir ad ʒiv. Sig. Teaspoonful every three or four hours during the day. Where there is profusion of venous piles below the sphincter—external hemorrhoids—*hamamelis* sometimes answers a better purpose, and two or three drachms of the distilled extract may be substituted for the collinsonia in the prescription just named. Where a local application is essential to relieve pain, the following may be employed: ℞ Fluid extract belladonna ʒii, oil of

erigeron canadense ʒiv, oleum olivæ ad ʒi. Sig. Apply morning and evening and at times of defecation. In stubborn cases *æsculus hippocastum* may be studied. Fullness, dryness and sense of constriction, with aching pain and weakness in the sacro-lumbar region is the accepted picture for its use, some asserting that it is specific, when there is absence of constipation. There are other remedies but these are the leading ones. When prolapsus attends defecation the use of a copious injection of weak salt water prior to the attempt at evacuation may assist in preventing this accident. Sometimes small doses of the 2x or 3x trituration of *podophyllin* may be used for this condition (prolapsus).

Dr. O. S. Laws (Dynamical Therapeutics) recommends a new remedy—*Negundium Americanum* (box elder)—above all others for specific action in hemorrhoids. He uses ten or fifteen grains of the powdered bark of the roots of yearling plants, or tablespoonful or more doses of a decoction several times daily. "Recent cases of hemorrhoids can be completely cured in this way and the old hard ones temporarily relieved." He considers it far superior to collinsonia in this place. Probably a saturated tincture of the bark could also be relied upon, and it would be a more stable form. It is hopeful that it will be supplied to our drug market.

After permanent tumors of large size become established and organized, internal medication can only modify unpleasant symptoms—more radical treatment must then be employed. The Whitehead operation will come into use when the entire lower inch of the rectum becomes a mass of hemorrhoidal tumors, though hypodermic injections of carbolized glycerine and olive oil will cure most cases, when patiently tried. The sphincter should be stretched to prevent strangulation of the prolapsed mass after a treatment of this kind, though if care be exercised this may be avoided by putting the tumor up after an operation, and the divulsion may be allowed to go until the latter part of the treatment.

The *diet* should be spare and devoid of indigestible substances, and so selected as to encourage relaxation of the bowels.

Amyloid Degeneration of the Bowels.

This is a disease of rare occurrence, and when it appears it is usually secondary to phthisis or chronic suppuration, especially long-standing suppuration of bone. The degenerative action begins in the terminal branches of the mesenteric arteries and involves the intestinal wall later, sometimes affecting the entire thickness of the part, and being marked by ulceration of the mucous membrane.

Both the large and small intestine may be involved, though the lower portion of the ileum is most susceptible.

The *symptoms* are obscure, there being a painless, chronic diarrhœa, without local tenderness or fever, with gradual loss of strength. If there is ulceration of the mucous membrane there will be blood and pus in the stools, and such symptoms attended by phthisis, chronic suppuration, or other wasting disease may be interpreted as belonging to the condition under consideration. An autopsy will usually disclose accompanying amyloid degeneration of the liver and spleen.

Nothing but palliative *treatment*, on general principles, can be suggested, as fatal results sooner or later follow.

Diarrhœa.

Definition.—The term diarrhœa literally signifies "I flow," and is applied to a condition in which there is a frequent discharge of fluid or semi-fluid fæces, unattended by tenesmus.

Etiology.—Diarrhœa may be irritative, symptomatic, mechanical, nervous, choleraic, vicarious, critical, colliquative or functional in character. It attends many diseases under one of these guises, and sometimes exists as an independent affection due to functional disturbance of the alimentary canal or of the digestive processes.

Irritative diarrhœa attends all cases marked by inflammatory invasion of the intestinal mucous membrane, such as the various forms of enteritis, typhoid fever, certain cases of mineral poisoning, intestinal worms, excessive biliary discharges, drastic catharsis, improper food, etc.

Symptomatic diarrhœa may be the result of certain acute and chronic affections, such as Bright's disease, the exanthemata, pyæmia, leukæmia, etc.

Mechanical diarrhœa is the result of obstruction to the portal circulation, causing transudation of serum from the bloodvessels into the intestinal canal. This may occur in hepatic, cardiac or pulmonary affections.

Nervous diarrhœa may be due to grief, great anxiety, fright or severe shock or pain. The discharges are then largely serous, though if the exciting cause appear soon after eating there may be evacuation of undigested food (lienteric diarrhœa).

Choleraic diarrhœa is the term applied to the watery evacuations which pass with a gush, during cholera, cholera morbus and cholera infantum.

Vicarious diarrhœa is the result of sudden arrest of secretion, usually of the skin, the diarrhœa being compensatory. It may fol-

low sudden chilling of the surface, or may attend undue indulgence in diet during hot summer weather. In other cases, it may be due to overeating.

Symptoms.—Under this head several divisions of diarrhœa have been suggested. In simple *fæcal diarrhœa* the evacuations are normal as to character, but increased in quantity and fluidity. In *bilious diarrhœa* the discharges are greenish-yellow, suggesting an abnormal amount of bile, though bismuth or other drugs may cause similar appearances, and due allowance should be made for medication to which the patient may have been previously subjected. When the evacuations are largely water the condition is termed *serous diarrhœa*, while mucous and muco-purulent evacuations may afford *mucous diarrhœa*. *Fatty diarrhœa* may attend faulty pancreatic action, and *crapulous diarrhœa* may follow immediately upon overindulgence at the table. A *critical diarrhœa* may attend the crisis of a disease, disappearing after the crisis is over, and a *colliquative diarrhœa* (profuse and serous) may attend the close of such wasting diseases as Bright's disease, phthisis, cancer, etc.

Frequent large evacuations mark an attack of diarrhœa, though the size of the discharges diminishes as the disease continues. The evacuations are often expelled with a gush, especially if the discharges be watery in character, though in other cases they may not be forcible. Serous diarrhœa is often attended by cramps in the extremities, and colicky pains and the expulsion of flatus may occur, though in some cases it may be painless. Febrile action may attend some cases, and thirst, chilliness and anorexia be present. Serous diarrhœa is usually attended by scanty urinary secretion, and the urine may be albuminous and highly acid. Large quantities of free fat may be found in the stools of fatty diarrhœa. Some cases of diarrhœa may result beneficially, though usually rapid prostration attends protracted cases. Digestion and assimilation are interfered with, and the patient loses flesh rapidly, the skin becoming dry and harsh, and the individual irritable and despondent.

Chronic diarrhœa is dependent upon some structural disease of the alimentary canal, such as chronic enteritis, intestinal ulceration, tubercular or syphilitic disease of the intestines, scurvy, malaria, etc.

Treatment.—The treatment of diarrhœa will depend upon the cause and the condition of the patient at the the time of attack. If the diarrhœa depend upon the presence of irritating food some unirritating but active *cathartic*, such as the compound powder of jalap and senna, may be used to assist in its removal. Following this a *soothing agent*, such as kaki, epilobium, bismuth, rhus aromatica or other remedy, should be administered, in appropriate doses. Sometimes potassium bichrom. 3x will serve a better purpose. When

serous diarrhœa is present, two possibilities as to cause are presented, namely, obstruction to the portal circulation, and relaxation of the intestinal capillaries. In the first attention must be paid to the hepatic, cardiac and pulmonary circulation, and in the other *capillary astringents*, such as erigeron, cinnamon, achillea or other agent, should be employed. In bilious diarrhœa chelidonium, bryonia, podophyllin in minute doses and, in some cases, mercurius dulcis 3x may be thought of. Oxide of zinc has been highly recommended in nervous diarrhœa. Fatty diarrhœa is best treated with olive oil and a diet composed largely of pickled olives. A wineglassful of olive oil should be taken three or four times daily, and pickled olives taken freely with each meal. Lienteric diarrhœa may require the use of ipecac, aconite, rhus tox. or other sedative. In all cases fermentation should be prevented by the frequent use of half-drachm doses of Marchand's *hydrozone*, diluted at the time of administration with two ounces of distilled or boiled water. Malarial diarrhœa will call for arseniate of quinia 3x, quinine or other antimalarial agent. A bland diet and the recumbent position are important.

Constipation.

Definition.—Constipation is a term applied properly to retention of fæces from any cause, though here it is intended to refer to a condition where the retention is due to functional derangement of the bowels and where the obstruction is purely fæcal in character.

Etiology.—Habits and modes of life often give rise to habitual constipation. Among these may be mentioned sedentary habits, neglect to attend promptly to daily evacuation, habitual use of opiates and alcohol, sparing ingestion of fluids, etc. Hepatic torpor may be provocative of it, many dark persons suffering habitual constipation all their lives from this cause. Certain chronic diseases, as neurasthenia, anæmia, hysteria and structural derangement of the brain and spinal cord may cause it. Uterine affections attended by irritation and congestion are common causes of the condition among women, and prostatic hypertrophy among old men frequently results in it. Certain articles of diet may cause fæcal impaction at first, and if this condition persists for a long time a constipation habit is finally established, through permanent perversion of the intestinal secretions. Atony of the bowels may be due to long-continued distention, and when this condition is relieved the intestinal torpor may be so established as to demand vigorous measures for its permanent cure. Diseased conditions of the mucosa may result in persistent dryness in some portion of the intestinal canal, fæcal accumulation stubbornly occurring there. Prolonged mental labor, melancholia,

insanity and other disturbances of the brain are not unlikely to be attended by constipation. However, by far the most common cause and one which gives rise to conditions just suggested as etiological factors, is orificial irritation—rectal pockets, papillæ, fissures of the anus, hemorrhoids, etc.

Pathology.—Though no lesion may appear at first, long impaction of fæces may result in dilatation of the intestine, with thickening of its walls. Pressure and irritation of hardened fæces may give rise to ulceration of the mucous membrane, and perforation sometimes occurs, both from ulceration and increased peristaltic action. The dilatation which occurs most markedly about the sigmoid flexure may result in paralysis of the muscular coat of the intestine, and pouches containing fæcal material and mucus may form along the colon. The impacted fæces sometimes become so hardened as to resist the edge of a knife. Various accidental substances, such as the stones of various fruits, hair, pebbles, gall-stones, etc., may form the nuclei of such impactions, and though not formidable enough to cause obstruction by themselves, they may assist in the formation of masses which may resist all efforts at dislodgment. Such diseases as hemorrhoids, rectal abscess, fistula, etc., may complicate cases of severe and long-continued constipation, and be the result, as well as the cause of them.

Symptoms.—The symptoms of constipation are so varied that the only one that can be relied upon is absence of regular fæcal evacuation, without severe local and constitutional symptoms. Subjects of the disease are liable to be dyspeptics, to have headaches, erratic appetites, insomnia, and be melancholic and despondent. Sympathetic disturbance of the hepatic functions, with slight symptoms of jaundice, is liable to attend. Periodical migraine and colic are common with such individuals. After long continuance the skin becomes torpid, dry and scaly or shriveled, the secretions generally rank and offensive, and the breath fœtid. Cardiac palpitation occurs at intervals, and there is often pectoral pain or aching under the scapula. Where the colon is distended pain in the region of the distention is almost a constant symptom, this usually being of a dull, aching character, though there may periods of acute aggravation. Neuralgic pains in the testicles, groins, down the thighs and in the lumbar region may be referable to impaction of the lower portion of the colon. Sometimes symptoms of intestinal obstruction may occur, and vomiting and cramping in the abdomen and other grave features appear. In many cases the bowels will move with difficulty every three or four days, the fæces being tenacious and pasty in character or consisting of hardened lumps which are evacuated with pain. Sometimes diarrhœa is alternated with constipation, the irritation of

the hardened fæces giving rise to periodical catarrh of the intestinal mucous membrane. Tunneling of hardened fæces may sometimes occur and the impaction persist, while fæcal material from above is passed through the hardened ring. Impaction may be detected by palpation, the fæcal accumulations forming large nodulated masses which are located along the course of the large intestine, forming movable tumors when located in the colon. When impaction exists near the sigmoid flexure tenesmus may attend, without power to evacuate the mass.

Diagnosis.—Constipation is a chronic condition hardly likely to be mistaken for intestinal obstruction, as that comes on suddenly unless it depends upon organic stricture of malignant or syphilitic character. Fæcal impaction may be attended by severe pain and vomiting similar to the symptoms of intestinal obstruction, but when it occurs it is not so likely to be persistent and severe. In such cases there is apt to be a history of long-continued constipation leading up to it.

Prognosis.—Functional retention of fæces is almost always amenable to treatment, few cases persisting if rationally managed. In extreme old age it may be difficult to completely overcome the intestinal torpor, though by judicious treatment fæcal impaction may be broken up and the bowels kept in a fairly active state.

Treatment.—The habitual use of cathartics to overcome constipation is usually pernicious. Innervation of the alimentary canal, that peristalsis may be encouraged, should be aided by such means as morning and evening massage, the abdomen being kneaded and well slapped over the bare skin for five or more minutes on each occasion. Exercise should be promoted where sedentary habits are necessary to any occupation, and plenty of fluids should be taken, with avoidance of tea and coffee. A cup of hot water before breakfast, with a cup of weak cocoa at that meal, will assist in promoting daily evacuation, especially if attention be paid to regular and daily effort at stool. Where there is intestinal torpor a single drop of tincture or fluid extract of *nux vomica* in the morning, taken in a glass of cold or hot water, is useful. Copious enemata of strong salt water, employed just after breakfast or dinner, regularly every day, may be tried in stubborn cases. The use of galvanism with the *salt-water electrode* is the most effective agent known in permanently curing intestinal torpor. It should be employed once or twice a week for several months, and afterward once a fortnight for a few times, until its good effect becomes permanent.

The common cause of chronic constipation is orificial irritation. All bad cases should be subjected to a rigid examination, for the purpose of detecting and correcting such a condition. After this

has been accomplished the use of the salt-water electrode should follow, as has already been suggested. Fæcal impaction may sometimes be broken up by judicious manipulation of the hardened masses through the abdominal walls, frequent use being made, meantime, of the salt-water electrode with galvanism. Where impacted fæces accumulate in the rectum and cannot otherwise be dislodged, they should be broken up with the finger, while the patient is under general anæsthesia.

The *diet* should consist largely of vegetables, coarsely ground cereals, fruits, especially cooked fruits, and plenty of water. Much lean meat should be avoided, as well as eggs, milk, sweets, puddings, pastries, fried foods, condiments, rich gravies, curry, sauces, pickles, nuts, tea and all alcoholic liquors.

Intestinal Colic.

Definition.—Pain in the intestines of functional origin arising from spasmodic contraction of the muscular coats of the bowel.

Etiology.—Intestinal colic is a neurosis, due to hyperæsthesia of the nerves supplying the intestinal canal arising from some exciting influence, such as the presence of irritating secretions or indigestible substances in the alimentary tract, dilatation of some part by fæcal accumulation, gases, intestinal worms, congestion from cold, rheumatism, gout, or hyperæsthesia of the terminal nerves through the effect of systemic poisoning by lead, copper, or alcohol. Liability to it decreases with advancing age. Women are more subject to it than men.

Pathology.—No appreciable morbid changes occur.

Symptoms.—The attack is usually abrupt, though it may be preceded by flatulence, nausea, borborygmus, chilliness and irritability of temper. The pain is abrupt in its onset, and, though it may be continuous, is marked by excerbations, during which the patient is bent forward or rolls about in agony, with groanings and cries of pain. The abdominal muscles are now rigid, the bowels are knotted and the face is drawn. The patient seeks to find relief by pressure upon the abdomen, either with his hands or by lying on his face. The abdomen is not sensitive to pressure and there is no soreness after the subsidence of the attack. There is absence of fever, the pulse is small and feeble and the extremities are often cold, especially during the paroxysms. The kidneys may be disturbed, a great quantity of limpid urine being voided during the attack and there may even be vesical tenesmus. There is sometimes vomiting (bilious colic) and constipation is the general rule, hough diarrhœa may be present. If allowed to continue the dis-

ease will usually terminate in the escape of flatus, either alone or accompanied by diarrhœa. Symptoms of collapse may attend aggravated cases. In malarious districts the attacks may be periodical, returning as regularly as an attack of ague, lasting several hours and then disappearing, until the second or third day afterward. In rheumatic colic there are usually accompanying pains in other parts, with tendency to metastasis. Bilious colic is the form in which nausoa and vomiting are prominent symptoms. Vitiated secretions or gastric complications are responsible for this phase. The vomited matters are greenish or yellow. The tongue may be coated, and there may be slight fever, suggesting more or less constitutional disturbance. This form may be more persistent than ordinary flatulent colic, and jaundice may appear during its development, the hepatic symptoms persisting for several days. It is most common in malarious districts. Colic frequently arises from a rheumatic condition, in which case metastasis to other parts is likely to occur.

Colica pictonum or *lead colic* affects those who have been using lead in some avocation for a long time, such as painters, compositors, type founders, stereotypers or lead miners. Sometimes accidental poisoning may occur from the use of water which has been carried through a lead pipe or in some such way. The pain comes on gradually in this form of colic, the paroxysms being moderately severe at first, but increasing in severity and frequency until intense paroxysms follow each other with rapidity. The colic is located principally about the navel, and cramps of the extremities may attend the paroxysms. The abdomen is flattened and hardened, the intestines are knotted and rigid, and there is obstinate constipation, which resists the action of all ordinary cathartics. Relapses are easily excited by the least indiscretion in diet or exposure to changes of temperature. The patient is sallow, anæmic, more or less debilitated, and the pulse is slow. A distinctive feature of the case is a deep blue dotted line along the margins of the gums, formed by a combination of sulpheretted hydrogen arising from decomposing food with the lead in the circulation. There is often paralysis of the extensors of the forearm, causing the wrist to drop when the arm is extended, or optic neuritis resulting in amaurosis, and tendency to epileptic convulsions.

Copper colic differs from lead colic in that the pain is increased by pressure, there is diarrhœa of greenish evacuations instead of constipation, and the abdomen is distended instead of contracted. The line along the edge of the gums is purplish instead of blue, and the specific influence of copper upon the laryngeal and bronchial muscles is manifested by spasm of these organs attended by dysp-

nœa. In both lead and copper poisoning there are elements of chronicity not observed in flatulent colic.

Diagnosis.—Peritonitis will hardly be confounded with colic, as it is a disease attended by marked febrile reaction and a tense, wiry pulse, with tenderness on pressure, while the opposite is the case in colic, the pain being paroxysmal instead of steady as in peritonitis; and these peculiarities will distinguish the disease from all other abdominal complaints.

Prognosis.—The prognosis is almost universally favorable. Convulsions may terminate unfavorably in children, though such a result is rare, and rupture of the intestine may occur in exceptional cases from violent distention of gases.

Treatment.—A specific remedy in most cases of *flatulent* colic is *colocynth.* The second or third decimal dilution of the specific medicine may be used, half a traspoonful being added to four ounces of water and a teaspoonful of the mixture being administered every fifteen minutes until the pain ceases, which will usually be after the second or third dose. Many Eclectics prefer to employ the old remedy, *dioscorea;* and this is indeed good, the dose being ten or fifteen drops of the specific medicine in a little water every fifteen minutes, until relief follows.

In *rheumatic colic* full doses of a strong decoction of *cimicifuga* root is best, the decoction being administered in wineglassful doses every half-hour, until its full effects are exerted upon the system. An *alcoholic vapor* or *cabinet vapor bath* assists materially here in shortening the course of treatment. Where periodicity is marked and the attacks persistently return with regularity, the use of antiperiodic doses of *quinine* or other antiperiodic is eminently demanded. When constipation is present a decoction of *rhamnus cal.* is more reliable and speedier in action than cimicifuga in rheumatic colic. The decoction may be taken in wineglassful doses, repeated every hour until its cathartic action is developed; then at longer periods. Gouty colic depends upon a gradually acquired constitutional condition which requires long-continued treatment for the gouty habit, as suggested under that disease.

Lead colic demands, for relief, prompt and urgent measures, and rigid abstinence from avocations or surroundings which tend to further contaminate the system. The obstinate constipation must be relieved, and for this purpose it is useless to depend upon ordinary cathartics. *Croton oil* is about the only drug that will accomplish the purpose here, and it should be given cautiously, in small but oft-repeated doses, until the purpose is attained. One drop of croton oil and one grain of powdered opium may be combined, and a dose given every two hours until the desired action occurs. Valuable

assistance may be derived from the use of galvanism with the salt-water electrode in the lower bowel, though this is not necessary. *Dioscorea*, in combination with gelsemium (sp. med. dioscorea gtt. xv, sp. med. gelsemium gtt. x), is excellent to alternate with the croton oil to alleviate the severe pains. As soon as the bowels move the pain ceases for the time, and further treatment should be directed to the prevention of a recurring attack. The salt-water enema with galvanism should be employed to promote regular evacuations, and a *milk diet* should be used for weeks—until the lead has been removed from the system. This is supposed to be furthered by the action of iodide of potassium, which combines with the lead in the system to form a soluble lead salt, which may be removed by the kidneys. If the patient cannot abandon his avocation or remove from the influences of surroundings which expose him to possibility of contamination, he should try to prevent the entrance of the drug into the system. As this is liable to occur during eating, a small dose of diluted sulphuric acid should be tak n during or after meals, this uniting with the lead in the stomach to form an insoluble compound of the metal, which cannot enter the circulation.

Copper colic should be treated similarly to other forms, except that as there is diarrhœa there will be no call for cathartics. *Sulphur vapor baths* may assist in removing the copper from the system, and cabinet vapor baths will alleviate, to some extent, the severe pain. A milk diet should be adhered to for a long time after convalescence.

Estival Infantile Enteritis.

Synonym.—Summer Complaint of Children.

Etiology.—Infants are peculiarly liable to diarrhœal diseases during the hot months of summer and early fall, many perishing every season from different forms of enteritis. The death rate begins in May and gradually rises into July, when it curves downward through August and September. Three important factors operate to bring about this state of affairs, namely, the want of development of the digestive organs, the character of the food consumed, and the development of bacteria in the intestinal canal. Until the deciduary teeth are developed the salivary glands of infants are incapable of digesting starchy food, and artificially fed children—those most liable to be affected with summer diarrhœa—are very apt to receive such aliment, unless the mother or nurse has been well instructed upon the subject; and this is not apt to be the case among the ignorant and poor, the class of people most liable to suffer. Hot weather encourages fermentation—the development of bacteria—

which may prove provocative of serious intestinal disease. Milk or other food that is least tainted is almost sure to contain many varieties of microbes, which, when they develop within the alimentary canal, originate toxines which may prove rapidly destructive to life. Most cases of mortality from infantile diarrhœa occur between the ages of six and eighteen months, and a very large majority in artificially fed children; the percentage of babies fed exclusively at the mother's breast affected being insignificant. The stools of healthy nursing children contain numerous bacilli and micrococci which seem to thrive when an exclusive milk diet is used, and this without detriment to the host, milk diet seeming to be the provision under which they exist; one species, the bacterium lactis ærogenes, being supposed to subsist upon the sugar of milk, while it develops in the upper portion of the alimentary canal. Another prominent form is the bacterium coli commune, which develops in the lower intestine. Other forms are present in health, but when enteritis arises the number is greatly increased, the morbid products then developed probably acting as toxines, as infantile diarrhœa in most cases is evidently more than a local disease. In children of the poor in cities, where fresh milk is difficult to obtain and where artificial foods are largely used, is where infantile diarrhœa marks its greatest ravages, though bottle-fed babies in rural districts are also frequently affected. Pure air, containing a large amount of ozone, neutralizes to considerable extent the virulence of the seasonal influence. On the sea coast and in mountain altitudes the disease is much less common. Around San Francisco Bay there is almost a complete absence of summer complaint among children, and when it occurs it must be managed very badly if the disease do not prove readily amenable. Oakland, on San Francisco Bay, it seems to me, is a paradise for bottle-fed babies.

Pathology.—The mucous membrane of both large and small intestines is swollen and covered with catarrhal secretion, and the lymph-follicles are enlarged, filled with proliferating cells and, in protracted cases, ulcerated. Occasionally there is croupous exudation on the mucous membrane of the colon and lower ileum, and, in such cases, extensive ulceration may occur. Lesions of the nervous system are not common, though in fatal cases effusion often occurs prior to death. The spleen and lungs may be congested, though such complications are not common. The liver and mesenteric glands are often congested.

Three varieties of summer complaint are described, all being due to similar causes, and all presenting similar pathological conditions, though the symptoms are markedly at variance. They are (1) *acute dyspeptic diarrhœa*, (2) *cholera infantum* and (3) *acute entero-colitis*.

Acute Dyspeptic Diarrhœa.—This form of summer complaint is characterized by the presence of undigested foods and curds in the evacuations, which are more frequent than normal, and of abnormal color, being sometimes greenish, sometimes greenish-yellow, again grayish-yellow, and often of a variety of colors. The disease comes on gradually, the patient being peevish, craving food and manifesting restlessness at night. In other cases the onset may be abrupt, and there may be colicky pains, vomiting and rapid rise of temperature, until 104° or 105° F. is reached. Sometimes active determination of blood to the brain with convulsions marks the onset. After the initiatory symptoms the case may continue in the form of lienteric diarrhœa, with greenish, tenacious discharges of fæcal material, mixed with gas and undigested food, or it may finally merge into a case of cholera infantum or entero-colitis. Such attacks may occur in very young infants as the result of improperly prepared cow's milk, or in older infants as the result of the ingestion of starchy or farinaceous food, or unripe fruit. In this disease the stools are tenacious and pasty, with occasional mixture of serous fluid, mucus rarely being present. Though there may be griping pain at the time of evacuation, there is no tenesmus.

Cholera Infantum.—In this form of summer complaint there are profuse watery evacuations, which are expelled forcibly with a gush, and the vomiting, which usually attends, is projectile, the ejected material being also watery in character. The disease is not very common as compared with the number of other cases of summer complaint which occur, though it is the gravest form of the disease.

The *symptoms* are very much like those of cholera morbus in adults, though the disease is more likely to terminate fatally. There are simultaneous vomiting and purging in many instances, though at other times the vomiting may precede the purging. The stools may contain fæcal material and be offensive in odor at first, but they soon become watery and odorless, and the patient becomes rapidly prostrated. The extremities become cold, the skin wrinkled, cold and clammy, the nails blue, the countenance pinched and pallid and the tissues greatly shrunken. Though the surface is cold the rectal temperature varies from 102° to 107° F. and the pulse is rapid and thready. There is extreme thirst and restlessness, and the patient may scream with agony from severe cramping pain at the time of evacuation. Liquids, foods and medicines are ejected as soon as swallowed in many instances, even a teaspoonful of water exciting responsive vomiting. As the disease progresses cerebral symptoms may appear, the temperature becoming very high, and the patient may die in convulsions within a few hours. In other cases the vomiting and purging may cease and the child pass into a comatose con-

dition, in which state it may remain for several days without change, lying with the head retracted, with irregular respiration and convulsive symptoms (hydroencephalon).

Acute Entero-Colitis.—This form of summer complaint is marked by the frequent evacuation of dejections of mucus mixed with fæcal material, and often streaked with blood, the evacuations being attended by painful straining (tenesmus) and preceded by pains along the course of the colon or about the umbilicus. Frequently there is gastric irritability, the tongue being red at the tip and pointed, fluids and foods being rejected. This is the common form which estival infantile enteritis assumes, and it usually follows acute dyspeptic diarrhœa. The follicles of the ileum and colon are the anatomical parts most involved in pathological change, and the term "follicular enteritis" is occasionally applied to it. Though usually a disease of hot weather, this form of enteritis may occur at any time of the year. The symptoms vary according to the portion of the intestine affected, evacuation without marked tenesmus attending when the irritation is in the lower portion of the colon or in the rectum. After the disease becomes established the evacuations are almost entirely mucus and blood, while there is nearly complete arrest of fæcal evacuation. Sometimes the inflammation of the rectum and colon is so severe that, instead of streaks of blood in the mucous evacuations the discharges appear to be nearly all blood, and there is more than ordinarily severe pain and tenesmus at time of evacuation. Colicky pains about the navel precede and announce the time of evacuation. The temperature is not usually so high as in cholera infantum, but there is constant elevation of temperature, this sometimes assuming a periodical character and manifesting remissions and excerbations, the severity of the intestinal trouble corresponding with the periodicity. The number of evacuations vary from ten to thirty in twenty-four hours.

As the disease progresses the patient becomes peevish and fretful, and gradual emaciation ensues. When badly treated the disease may continue for weeks, the evacuations gradually becoming purulent and general wasting of the tissues (marasmus) attending, the skin becoming dry and wrinkled, the eyes sunken, the face pallid with hectic flush, and the child generally prostrated, with irritable stomach. Death from convulsions frequently closes the scene.

Treatment.—An important consideration in the treatment of any form of summer complaint of children is rest for the alimentary canal. As milk, otherwise than human breast milk, is almost certain to curdle in the stomach and remain, to considerable extent, undigested thereafter unless pancreatinized before administration, and as such food doubtless contains many causal elements of the disease (if

not the specific elements), it is a wise plan to withdraw it entirely for a time as a diet and substitute such food as cannot furnish any solid material during digestion to irritate the alimentary canal. *Barley-water* or *rice-water* may be substituted for the first thirty-six hours or longer, in children a year or more of age, these, to say the least, contributing no additional source of danger. Strict attention should be paid to the avoidance of fermentative action, all food being kept in a refrigerator, or sterilized each time before use. When the disease is severe and protracted, fresh meat broths may be substituted. In younger children the use of Horlick's malted milk (which is so combined with cereals as to prevent coagulation) may sometimes prove better than broths or barley-water, the food being carefully sterilized at each feeding, and the periods being regulated to the requirements of individual cases, and not allowed too often. *Eudoxine* or *glycozone* may assist in arresting fermentation.

The milk of pregnant mothers is sometimes very injurious, occasionally, apparently, being a provoking cause of the complaint. I have found it necessary to remove the child from the breast on this account, and in one case where death seemed imminent immediate improvement followed when Mellin's food was substituted for the mother's breast. When cow's milk is to be used, it should be carefully sterilized and afterward combined with lime-water, in order that the curd may be well broken up and acidity removed—a tablespoonful of lime-water to eight ounces of milk. Nothing but milk sugar should be used for sweetening the food of bottle-fed babies less than a year old.

The *medicinal treatment* of dyspeptic diarrhœa will consist of immediate evacuation of the bowels, the neutralizing cordial of the American Dispensatory being an excellent article for this purpose. Following this, one or two drops of *hydrozone* in a drachm of distilled water may be administered three or four times daily to neutralize fermentation and destroy toxic germs. If the greenish, tenacious stools persist after this, two-grain doses of mercurius dulcis may be administered every three or four hours until several doses have been administered, this remedy usually being capable of altering the conditions so that return to normal fæcal evacuations follows. Sometimes two- or three-drop doses of the 3x dilution of *mercurius cor.* will answer better, and at others five grains of *sodium sulph.* in four ounces of water, dose a teaspoonful every hour, will answer better. Minute doses of *sulpho-carbonate of zinc* are also excellent here.

In *cholera infantum* the hydrozone should be used as already suggested, and to arrest the gushing discharges and projectile vomiting half a drachm of *veratrum album*, 3x dilution, may be added to four ounces of distilled water and a teaspoonful administered every fif-

teen minutes or half-hour. If this fails to control the watery discharges, or if it is not immediately obtainable, a decoction of the fresh *erigeron canadense* plant may be allowed, the child here drinking it with avidity. It may be given often and freely. The specific medicine may answer as well, though I have never tried it for the purpose. When hydroencephaloid symptoms appear *aconite* and *rhus tox.* should be administered, two or three drops of Lloyd's aconite and eight or ten drops of rhus tox. being added to four ounces of water and a teaspoonful given every hour. Where symptoms of coma are marked and the pulse is small, feeble and compressible, two or three drops of specific *belladonna* may be used instead after the same manner as the aconite and rhus tox. In other words, two or three drops of specific belladonna may be added to a half glass of water, a teaspoonful of this to be given every hour until comatose symptoms subside.

In the treatment of *entero-colitis* quite a wide range of remedies may be required, though a few are usually sufficient. *Aconite* and *ipecac* answer, with proper feeding, in relieving most cases and perfecting a cure. Add two or three drops of aconite and ten or fifteen of specific ipecac to four ounces of water and order a teaspoonful every hour. Sometimes the abdominal pain is excessive and attracts special attention, demanding something more specific for its relief. Then we will administer *colocynth* 3x dilution, half a drachm in four ounces of water, a teaspoonful every hour or oftener if desired, until this phase is removed. Colocynth is especially desirable if there be much blood in the stools. In these cases time is an essential element of success. Several days may be required to effect a cure, the first favorable symptom being a lessening of the severity of the suffering during and between stools, and the next a dimunition in the number of stools. In malarious districts there will be a marked periodicity which will demand *arseniate of quinia* 3x, and this remedy may be used with good judgment in any case where malaria is liable to be present.

Sometimes there is evidence of necrotic tendency, the evacuations containing shreds of mucous membrane, false membrane and dark sanious discharges (prune-juice), and the patient evincing typhoid symptoms. Here we may think of *baptisia* or *echinacea*, the latter remedy being usually more appropriate, though sulpho-carbolate of sodium may often supersede other remedies in such instances.

Where the disease runs into a chronic form and the discharges become thin and watery with muco-purulent admixture, the patient thin and emaciated with wrinkled skin, constant fretfulness and other symptoms of marasmus, *mercurius cor.* 6x is an excellent remedy for its relief. It is also excellent in the early stages, where the stools

are greenish in color. In marasmus following this condition an almost indispensable element of rapid and satisfactory success is the *tonic faradic* treatment, general arrest of all unpleasant symptoms rapidly following its adoption.

Several other important remedies for this disease and cholera infantum may be found in Dynamical Therapeutics, a work almost indispensable for the study of modern materia medica.

VIII. DISEASES OF THE MESENTERY.

Miscellaneous Affections.

The mesentery is liable to serious affections, which are of sufficient importance to merit attention in a work of this character.

The mesenteric *arteries* are subject to embolism and thrombosis, and when this occurs the bowel in the territory supplied by an affected vessel undergoes a condition of infarction. When only a small branch is affected the condition may not be serious, and restoration may occur without the development of serious symptoms. When larger vessels are blocked, however, severe pain in the abdomen, with tympanities, nausea and vomiting, soon arises. Diarrhœa occurs as a rule, the stools being thin and watery and sometimes tinged with blood. Thrombosis of the superior mesenteric artery is followed by infarction of nearly the entire length of the small bowel, and rapidly fatal conditions follow. The treatment can only be of a surgical nature, resection of the bowel being suggested. Where the infarction involves but a small section of the intestine resection offers hopes of relief.

The mesenteric *veins* are subject to dilatation, sacculation and calcification, distention being due to portal obstruction. This may occur in cirrhosis of the liver or in any other condition in which the onward motion of the blood toward the portal circulation is impeded.

Suppuration of the mesenteric veins is also liable to occur, and it is a usual attendant of inflammation of the vena portæ. Extreme dilatation of the mesenteric veins then follows, and large quantities of pus accumulate, until the mesentery appears, upon inspection, like a bag of pus. Upon careful examination, however, the pus will be found to be confined within venous channels which have undergone great dilatation. The symptoms resemble those of pylephlebitis, though there is greater abdominal distention. Treatment is of little avail.

Cysts of the mesentery are not of unfrequent occurrence. Quite

a variety of morbid formations of this character may occur here. They may be chylous, serous, sanguineous, hyatid or dermoid. They may vary, from the size of an orange to immense masses occupying and distending the entire abdomen. They may develop slowly or rapidly, from a few months to ten or twelve years being the varying time occupied in their growth in different cases. The general health is not much affected in these cases, colicky pains and constipation, with enlargement of the abdomen, being the principal symptoms. The diagnosis is obscure, such diseases as ovarian tumor, floating kidney, hydronephrosis and omental cysts being liable to be confounded with it. No specific treatment is known.

The *chyle vessels* are subject to various morbid conditions. Enlargement of the ducts in the mucous and submucous tissues of the intestine and stomach occasionally occur. Sometimes these are cystic, sometimes varicose, and sometimes cavernous. Extravasation of chyle into the tissues of the mesentery sometimes occurs.

IX. DISEASES OF THE LIVER.

Jaundice.

Synonym.—Icterus.

Definition.—Jaundice can hardly be classed as a distinct disease, as it may depend upon a variety of pathological conditions. However, as it is a striking condition of the system frequently present, a consideration of the various phases attending it under one grouping will be not only proper, but essential. The term "jaundice" or "icterus" belongs to conditions of the system marked by the presence of bilirubin in the general circulation and in certain of the secretions, such as the urine and perspiration, and characterized by yellow hue of the skin, conjunctiva, hard palate, etc.

Etiology.—Two forms of jaundice occur, one being due to obstruction of the bile-ducts and the other to imperfect service of the hepatic cells. When the bile is secreted by the hepatic cells but is retained in the biliary ducts, to be afterward absorbed by the lymphatics and carried to the blood through the thoracic duct, the condition is termed "hepatogenous or obstructive jaundice." This may be due to tumefaction of the mucous membrane lining the biliary ducts, especially of the common bile-duct—that portion which lies in the wall of the duodenum—the condition often being the result of inward extension of congestion of the intestinal mucosa. At other times the obstruction may be due to lodgment of biliary calculi, or in exceptional instances to such parasites as the distoma

hepaticum or echinococcus in the common bile-duct, and again to pressure from tumors, such as cancer of the pylorus, tumors of the pancreas, liver, or other abdominal tumors. The gravid uterus may exert such pressure, as well as aneurisms. Inspissated mucus may obstruct the opening of the biliary ducts where there is catarrh of their lining membrane, it being remembered that the bile-pressure from the secretory action of the cells in the ducts is very low, and that the lymphatics readily take it up if there does not exist a favorable way of exit.

When there is lack of power upon the part of the biliary cells to separate the coloring material from the hepatic circulation the condition is termed "hematogenous or nonobstrusive jaundice." In this form there may be an excess of blood destruction, as in malaria, yellow and typhoid fever, epidemic jaundice, pyæmia, snake-poisoning and poisoning from phosphorus and other drugs, the hematoidin resulting being identical with the coloring material of the bile (bilirubin). Necrosis of the hepatic cells may prevent separation of this substance, as in case of acute yellow atrophy, yellow fever, or such other infectious diseases as pyæmia, etc.; or, the destruction of red blood-corpuscles may be so extensive that an overflow occurs, and, while a normal amount may be separated, a sufficient quantity appears in the general circulation to give rise to a jaundiced condition of the tissues.

In icterus neonatorum or the jaundice in infants, the first few days succeeding birth may be attended by jaundice due to effective closure of the ductus venosus, or to lack of pressure in the branches of the portal vein due to arrest of the placental circulation. In grave forms of infantile jaundice congenital syphilitic hepatitis may be a cause of obstruction, there may be congenital closure or absence of the biliary duct, or there may be septicæmic phlebitis of the hepatic veins from infection of the umbilical cord.

It is a question with many whether true jaundice can exist unless the hepatic cells have formed the bile previous to its admixture with the blood—whether such a disease as hematogenous jaundice can occur. While the coloring material of the bile is identical with hematoidin, which may be formed without passage through the liver, the bile-salts (glycocholate and taurocholate of soda) never exist unless elaborated by the hepatic cells. At least, then, there is a marked difference between hepatogenous and hematogenous jaundice in that in the one true bile circulates in the blood, while in the other only the coloring material, identical with that of bile, is present without other biliary constituents.

In Weil's disease (an acute infectious fever attended by jaundice, duly considered under specific infectious diseases) there is obstruc-

tion of the biliary ducts from swelling of the liver, the clay-colored stools attesting absence of the coloring material of the bile from the intestinal canal.

Pathology.—The morbid change which follows the continued presence of the bile in the blood in hepatogenous jaundice is effusion of bile-stained serum which yellows nearly all the tissues of the body. It is asserted that the humors of the eye and substance of the brain usually escape. Even the bones and teeth may be colored, as well as new pathological formations. The presence of the foreign material may be tolerated for a time, but *cholœmia* or *cholesterœmia* is finally likely to result, a poison being generated which sets up typhoid symptoms attended by fever and succeeded by coma, delirium or convulsions.

The diversified conditions attending various forms of hematogenous jaundice will be referred to under the special diseases in which they occur.

Symptoms.—The staining of the tissues is most marked in *hepatogenous jaundice*. The tint may vary from a lemon-yellow to a deep olive-green or bronze, the tint depending upon the permanency of the obstruction of the biliary ducts. As catarrhal jaundice usually terminates within a few days, the extreme depth of color reached in more serious obstruction, as where permanent organic change exists, does not here occur.

The skin and conjunctiva are markedly colored, the bright-red mucus membrane, such as that of the lips, tongue and buccal surfaces, not showing the stain to any great extent, though a distinct yellowness of the hard palate may, in some instances, be observed.

Of the secretions, those most deeply colored are the urine and perspiration. The color of the urine, in which the pigment may be found before it is apparent in the skin or conjunctiva, may vary from a light greenish-yellow to a deep black-green. A chemical test may be made for it by placing a few drops of the urine on a white porcelain plate and adding a drop or two of nitric acid, when, if bile be present, a rapid play of colors is produced, various shades of violet, yellow, green and red interchanging. The urine colors white linen yellow, and the perspiration, especially in the axillæ and groins, may stain the underclothing a similar color. In long-standing cases the urine may contain albumin and bile-stained tube-casts. The tears, saliva and milk are not usually stained, although the expectoration may be colored when inflammatory action in the pulmonary tissues exists along with jaundice. Usually, however, the sputum is not affected. Arrest of the usual flow of bile into the intestine is attested by clay-colored, fœtid stools, constipation or diarrhœa.

Pruritis of the skin is a frequent symptom of long-standing cases, and it may be present in brief catarrhal jaundice, though it is not apt to appear until the condition has become somewhat protracted. Various eruptions may develop upon the skin, such as boils, wheals, lichen and urticaria. Sweating of the abdomen and palms of the hands is sometimes a persistent symptom.

The pulse is often markedly reduced in frequency in obstructive jaundice, the action of the heart falling to forty, thirty and even twenty pulsations per minute. It is not considered a serious symptom, however, being probably due to temporary impression of the biliary material upon the cardiac nerves.

The effects of the biliary element upon the nervous system are variously manifested. Drowsiness is frequently present, jaundiced subjects being inclined to lethargy and sleep. During waking hours, irritability and melancholia may be marked. Delirium, coma or convulsions are liable to develop suddenly in any case of protracted jaundice, and typhoid symptoms frequently terminate such cases. Such cases are more apt to attend hematogenous than hepatogenous jaundice, however, though long-continued presence of bile in the blood in the latter form is liable to at length develop cholæmia or cholesteræmia, with grave symptoms.

In hematogenous jaundice there is not so marked discoloration of the skin as in obstructive jaundice, and febrile symptoms with rapid pulse are common. Bile-pigment is not so common in the urine, though the urinary pigments may be increased, and the stools are not clay-colored as in the obstructive form. Cerebral symptoms are more liable to be marked here than in hepatogenous jaundice, toxic forms being marked by delirium, coma, convulsions and speedy demise.

The *treatment* of jaundice, in its various forms, will be considered under the different diseases giving rise to it. In some cases treatment is effective and highly satisfactory in its results, while in others, on account of the pathological changes present, even temporary relief is impossible.

Infantile Jaundice.

Synonym.—Icterus Neonatorum.

Etiology.—The causes of infantile jaundice have already been referred to. Reduction of blood-pressure in the hepatic capillaries due to arrest of the umbilical circulation may prevent proper action of the hepatic cells for a brief time, or temporary communication between the portal and general circulation may account for mild cases which recover spontaneously. In the severe forms there may

be congenital closure or absence of the common bile-duct, hepatic syphilis of congenital form, or phlebitis from septicæmic infection of the remains of the veins in the stump of the umbilical cord.

Symptoms.—It is frequently the case that new-born children become jaundiced within the first two or three days of life and incline to drowse continually. The skin presents a deep, yellowish-red color, instead of the reddish tint usually observed. The urine stains the diapers yellow and the fæces, after the passage of the meconium, are colorless. The child may nurse, however, digest its food fairly well and not manifest any symptoms of distress. The well-meaning but misinformed nurse may now administer a decoction of saffron to "clear up the skin," and in a few days the jaundice disappears—a result which nature would have accomplished as well without the "saffron tea" as with it.

Infantile jaundice from atresia of the bile-ducts is a rare condition, though several children of the same parents have been known to be similarly affected. The attendant jaundice may not be appreciable to sight for a week or fortnight, or even more. The skin, conjunctiva and hard palate become yellow, and the tint rapidly grows darker. The liver enlarges, the abdomen becoming protuberant and distended, the swelling being largely due to hepatic and splenic congestion, though intestinal gases and ascitic fluid may contribute to the enlargement. Swelling of the hemorrhoidal veins and bleeding from the navel may attend, the latter symptom being especially noticeable, often beginning soon after the fall of the navel-string and continuing to ooze until death, the discharge probably being due to obstruction of the portal circulation from the swelling of the liver. When umbilical hemorrhage is combined with infantile jaundice from atresia, death follows within a few days, though jaundice from congenital atresia may otherwise continue for several months before a fatal termination, the child taking food well, but gradually wasting away, death possibly occurring finally from some accidental complication, such as bronchitis or pneumonia.

Where infantile jaundice is due to syphilitic inflammation of the liver, there are such suggestions of syphilis as skin eruptions, snuffles, etc. The jaundice appears at birth, the liver is much enlarged, and there is bleeding from the umbilicus and bowels, and extravasation into the skin. Rapid wasting and loss of strength are followed by subnormal temperature, convulsions and death.

When the jaundice is due to umbilical phlebitis the yellow discoloration of the skin comes on a few days after birth and is attended by fever, vomiting and complete loss of appetite. The child refuses the breast, appears pinched and haggard, the tongue becomes dry, and the hands and feet purple. The abdomen swells rapidly

and is tender upon pressure, and there is more or less distinct fluctuation, while blood and sanious pus ooze from the navel. The jaundice is marked, and the urine may be intensely yellow, though the stools may not be affected. Convulsions or coma may precede death.

Treatment.—Little benefit can be expected from treatment. In mild infantile jaundice minute doses of *chionanthus* may sometimes assist in removing the coloring material from the circulation, though probably a safer plan would be to leave the case to the unassisted efforts of nature. In the jaundice from atresia and syphilis nothing can be expected, though *echinacea* and *lachesis* may be thought of in umbilical phlebitis.

Malignant Jaundice.

Synonyms.—Icterus Gravis; Acute Yellow Atrophy of the Liver.

Definition.—A grave form of jaundice characterized by cerebral symptoms and distinguished by extensive destruction of the cells of the liver, with the deposit of leucin and tyrosin in the urine. Extensive necrosis of the liver-cells is attended by marked reduction in the size of the organ.

Etiology.—This is a rare disease, and one which seems more common in Europe than in this country. The rapid and extensive necrosis which affects the hepatic cells suggests a powerfully toxic influence which can hardly be ascribed to any other cause than that of bacterial origin. Pregnant women seem to be especially susceptible to it, as quite a large proportion of those affected have been of this class, though males are also subject. A majority of cases occurs in individuals between twenty and thirty years of age, though it may affect children. It is said to have followed sudden fright or profound thought, excesses in venery and poor living.

Pathology.—The liver is remarkably reduced in size in most cases, a thinning of the organ being a feature. When cut the surface is yellowish-brown or reddish-brown, and microscopical examination discovers more or less complete destruction of the hepatic cells through the entire extent of the organ. Complete destruction of the cells may be discovered in some places, while partially destroyed structures remain in others. Granular débris, containing pigment and crystals of leucin and tyrosin, occupy the devastated sites. The capsule of the organ is wrinkled, the bulk having shrunken to a half or a third of its original size and weight. Microörganisms have been found in the liver-tissues by various observers. The kidneys are liable to be involved, granular degeneration of the

epithelium occurring. The spleen is enlarged, and the heart is apt to undergo fatty degeneration. Various organs are stained with bile and extravasated with blood. The bile-ducts and gall-bladder are empty.

Symptoms.—The symptoms may not be severe in the beginning, the condition resembling at first a mild case of obstructive jaundice complicated with gastro-duodenal catarrh. Continuing in this way for from a few days to two or three weeks, a period arrives at which all the symptoms become suddenly aggravated. There is vomiting, persistent and constant; frequent hematemesis; and hemorrhaghes may occur into the skin, conjunctiva and other parts. Nervous symptoms are now a marked feature, there being intense headache, trembling of the muscles, often delirium, and even convulsions. A marked increase of the icteric symptoms attends this aggravation, febrile conditions are assumed, the temperature rises, the pulse becomes rapid, and typhoid symptoms, such as dryness of the tongue with brown coating and sordes on the teeth and lips, and muttering delirium or coma follow. However, pyrexia is not always present.

The stools are clay-colored, showing that no bile enters the intestine, and the urine contains bile, tube-casts, leucin and tyrosin.

Diagnosis.—Jaundice, with delirium and diminution of the size of the liver, suggests the presence of this disease. Delirium may attend hypertrophic cirrhosis, but enlargement of the liver will there serve to distinguish it, and febrile symptoms are more constant. Phosphorus poisoning may simulate this disease, as there are jaundice, hypertrophy of the liver and purpura; but leucin and tyrosin are absent from the urine and the gastric symptoms are more constant from the start.

Prognosis.—The disease is usually fatal under old-school treatment, and Eclectics have recorded little experience with it.

Treatment.—Goss advises minute doses of *aconite* and *ipecac* to control the vomiting and hæmatemesis, and *chionanthus* and *berberis vulgaris* for the biliary symptoms. Behind the symptoms, however, lies an important pathological change—destruction of the liver-cells by necrosis—which demands first attention. A small group of remedies gives us positive effects in many similar conditions, and may be relied upon here with good prospects, if begun early. This group comprises such remedies as *echinacea*, *baptisia* and *lachesis*. If the disease is diagnosed early, *potassium chloride* 3x, as usually employed, may prove a serviceable remedy. An easily digested liquid *diet* should be adhered to, such articles as peptonized milk, meat broths, buttermilk, clam broth and Horlick's malted milk constituting its basis.

Abnormalities of the Hepatic Circulation.

Active Hyperæmia.—This may follow meals as a physiological act, the rapid absorption of the portal vessels resulting in vascular fullness of the liver. When overeating is habitually indulged in, a condition of chronic hyperæmia may finally follow, with functional disturbance; and when alcohol is indulged in to large extent cirrhotic changes may arise. The principal *symptom* is a sense of fullness in the right hypochondriac region, with dypsnœa dependent upon difficulty in drawing the diaphragm downward, this passing off after time of digestion has gone by. Regulation of the diet is obviously the most important part of *treatment*, though *aploppapus laricifolius* will afford temporary relief (saturated tincture, gtt. v–x, in a swallow of water at a single dose).

Passive Congestion.—This involves the sublobular branches of the hepatic veins, and is due to backward pressure from the general circulation. Obstruction in the right heart or lungs, and tricuspid insufficiency may be causes—any condition attended by venous stasis in the right side of the heart, emphysema, pulmonary cirrhosis, intrathoracic tumors or chronic valvular disease being prominent among the clinical causes. Chronic pressure from cardiac impact may give rise to gradual dilatation of the sub-lobular and intra-lobular vessels, the intra-lobular capillaries compressing the hepatic cells until they may finally become atrophied, a cut section of the organ presenting a mottled appearance due to the large amount of venous blood in the central capillaries, deposit of pigment and augmentation of connective tissue; the condition being termed "nutmeg liver." Among the *symptoms* may be pulsation of the liver due to impact from the cardiac systole, gastro-intestinal catarrh, with occasional hæmatemesis. Ascites may finally appear as a result of obstruction to the portal circulation, and icteric staining of the skin attends, with bile-pigment in the urine and clay-colored stools. The liver is appreciably enlarged, the organ being crowded downward and rolling outward beneath the ribs, imparting, upon palpation, a sensation of firmness to pressure.

Treatment is not very satisfactory, as the disease is secondary, usually, to organic change of the heart or lungs, and a cure must depend upon a removal of the exciting cause. To lessen pressure in the portal vein, such remedies as *carduus marianus*, *polymnia*, *ceanothus* and *grindelia squarrosa* should be thought of. *Aploppapus laricifolius* is an excellent remedy in some cases for this purpose. The withdrawal of fifteen or twenty ounces of blood from the liver by aspiration has been recommended, but the result must necessarily be temporary, and would hardly, it seems, justify such a procedure.

Depletion of the portal circulation might be brought about by the free administration of hydragogue cathartics, though the gastro-intestinal catarrh would contraindicate the use of irritants here, the salines being more appropriate. Attention must be paid to the condition of the pulmonary and cardiac circulation, appropriate remedies being directed to abnormal states in these parts.

Diseases of the Portal Vein.—Chronic portal obstruction may arise from chronic congestion of the liver, the etiology of which has already been considered. Local causes are cirrhosis, pressure from tumors involving the liver or located in the vicinity of the portal vein, compression from proliferative peritonitis, or from thrombosis.

Thrombosis or adhesive pylephlebitis of the portal vein occurs secondarily, from pressure upon the portal vein or one of its branches from tumors, perforation of the vein by gall-stone or invasion by cancer. When it occurs primarily it is during moribund processes, and is not of importance. When occurring secondarily, the clot becomes organized, grows pale and firm, and may finally become converted into connective tissue. It may become perforated and permit of the passage of a limited quantity of blood, a permanent narrowing remaining and modifying the attending symptoms.

The *symptoms* are announced by vomiting with diarrhœa, usually attended by hæmatemesis and enterorrhagia. Dilatation of the portal vein behind the point of obstruction rapidly follows, with rapidly accumulating ascites. Dyspnœa, anorexia, prostration and heart failure result, a fatal termination usually following within a week or ten days. When some small branch only is involved, a collateral circulation may be established and the patient may live for years, though in indifferent health, general emaciation and prostration with occasional gastric or intestinal hemorrhage attending. The *diagnosis* is difficult, and the *prognosis* is exceedingly bad. *Treatment* is unsatisfactory, temporary relief, in all cases in which collateral circulation is not established, being all that can be expected. The ascitic accumulation should be removed by paracentsis, and the diet should be sparing. Diuretics and cathartics are recommended, but cathartics would be liable to provoke more discomfort than they would assuage. Restriction of the diet to almost a point of starvation would be the most rational measure to pursue.

Septic thrombosis or suppurative pylephlebitis of the portal vein is characterized by the formation of a thrombus in the portal vein with subsequent breaking down, from the presence of infective material. In this case the thrombus is not due to pressure, but arises from localized inflammation of the vein, caused by intestinal ulceration, abdominal abscess, or such penetrating bodies as spiculæ of

bone, pins, needles, tacks, etc., which have been accidentally swallowed and which penetrate the intestine, and later the portal vein. In infants septic material may enter from the way of the navel, and give rise to similar conditions. A clot forms at the point of penetration, to afterward break down from suppurative action. Emboli may be distributed to the liver from here, septic abscesses be scattered through its substance, and even general pyæmia may attend, the emboli sometimes passing the lobular circulation. Symptoms of portal obstruction, septicæmia, pyæmia, and multiple abscesses of the liver occur. Fatal results invariably attend within ten days or two weeks, and treatment can be palliative only.

Affections of the hepatic vein are rare, its peculiar structure serving to protect it greatly against infection or embolus from the abdominal circulation. Enlargement of the right heart results in its dilatation, and stenosis may arise at the orifices of its branches, general enlargement and induration of the liver being the result.

Dilatation of the hepatic artery may attend cirrhosis of the liver, and it may be sclerosed, or be the seat of aneurism.

Interstitial Hepatitis.

Synonyms.—Cirrhosis of the Liver; Sclerosis of the Liver; Gin-drinker's Liver; Hob-nailed Liver. The term "cirrhosis" was applied by Laënnec, on account of the yellow color of the diseased organ.

Definition.—An inflammation of the connective-tissue of the liver, attended by strangulation of the hepatic circulation, and consequent destruction of the hepatic cells.

Etiology.—Influences which originate and perpetuate irritation of the capillaries of the hepatic circulation predispose to this condition. The most common is probably alcoholic addiction, the habit of taking spirituous liquors on an empty stomach being especially liable to produce it, as the stimulating influence of the alcohol on the hepatic circulation is then most pronounced. Highly seasoned food containing stimulating condiments is liable to result in a similar condition, when indulged in for a long time. The acute infectious diseases, notably scarlet fever, may inaugurate interstitial hepatitis. Gout, syphilis and rheumatism may be included among the predisposing causes. Malaria, by producing continued engorgement of the portal circulation, is not an infrequent cause. Cardiac and pulmonary obstruction may be attended by sufficient backward hepatic impact to result in final cirrhosis. It often attends tuberculosis of the liver. Anthracosis of the liver is said to be a cause among miners and workers in coal.

Pathology.—The connective tissue surrounding the smaller twigs of the portal vein is usually first involved, the inflammation gradually extending to the larger branches. The rapid proliferation of embryonic cells results at first in a soft, reddened, pulpy mass, which distends the portal canals and increases the volume of the entire liver. As the new cells undergo organization into permanent fibrous material contraction follows, and compression is exerted upon the portal, interlobular and central vessels, arresting their functions. Nutrition of the lobules is thus cut off, and the pressure exerted encroaches upon the hepatic cells, aiding in causing atropy and degeneration of their structure. The outer cells of the lobules undergo fatty degeneration at first, though complete obliteration of the lobules may follow, their places being filled, in some instances, with newly-formed connective tissue. The cells surrounding the central vein are degenerated, atrophic and deeply stained with bile. The portal canals present, upon the surface of the liver, depressions from contraction of fibrous tissue, the intervening lobules imparting a granulated impression, and, as the contraction proceeds, the entire organ may become corrugated and nodular upon the surface, affording the condition known as hob-nailed.

If a section of the organ be made during the early stage of the disease, the cut surface presents a hyperæmic, pulpy appearance and the entire organ is enlarged; but in a later stage the tissues are firm and fibrous, cut with resistance, and the section presents a mottled, yellow surface, upon which may be seen yellow spots stained with bile-pigment, representing the central portions of lobules surrounded by lighter colored zones of fatty degeneration, with surrounding areas of slaty-gray, fibrous material. The smaller portal vessels are shrunken, convoluted and twisted, and their lumen may be completely obstructed, new channels sometimes being formed between the portal and hepatic veins. Separate branches from the hepatic artery are sometimes traceable in the newly-formed connective tissue, and the main artery is dilated and tortuous.

In hypertrophic (fatty) cirrhosis, the new connective tissue insinuates itself about the bile-ducts and within the lobules, imparting a firmness which occurs coincidentally with the development of connective tissue in the portal channels, and resists contraction, the volume of the organ being permanently augmented by the growth of connective tissue in its minute structure and the deposition of fat in the parenchyma of the lobules. Another form of hypertrophic cirrhosis (biliary cirrhosis) is marked by early obstruction of the small biliary-ducts and their radicles, these becoming permanently distended and infiltrated with bile-pigment. Coincidently with this there is an abundant development of new connective tissue, but the

biliary engorgement offers an obstacle to contraction, and the organ not only retains its size but becomes augmented in bulk.

Syphilitic cirrhosis may result in atrophy or hypertrophy of the liver. When the capsule is largely involved, contraction of the organ results, and it may be diminished in size, the general anatomical condition resembling that of cirrhosis from other causes. In diffuse syphilitic hepatitis there may be permanent enlargement of the organ, large bands of puckered fibrous tissue, visible to the naked eye, appearing in isolated patches, and gummata being more or less numerous, scattered through the substance, the fresh gummata presenting a reddish-gray, translucent appearance, and the older ones being surrounded by connective-tissue capsules, their centers being broken down into puriform material or transformed into fibrous, cheesy or calcerous masses.

Anatomically, four forms of hepatic cirrhosis have been recognized: (1) The atrophic cirrhosis of Laënnec; (2) perihepatitis or Glissonian cirrhosis; (3) fatty cirrhosis; and (4) hypertrophic cirrhosis. Two of these forms, viz., the atrophic cirrhosis of Laënnec and Glissonian cirrhosis, are attended by final atrophy, and two, fatty cirrhosis and hypertrophic cirrhosis, are attended by permanent enlargement.

In the *atrophic cirrhosis of Laënnec* the liver, in advanced stages, is very much diminished in size, and its tissues are remarkably firm and resistant to the knife when cut. Its outer surface is granulated, the contraction which it has undergone may have resulted in deformity of shape, and its weight may be reduced to a third or a fourth of its normal amount. When the cut surface is examined critically, it will be seen to present isolated, greenish-yellow spots, surrounded by grayish-white fibrous tissue.

In *Glissonian cirrhosis* there is remarkable development and fibrous degeneration of Glisson's capsule, due to localized peritonitis involving the perihepatic membrane. The capsule is hardened, almost cartilaginous in consistency, and adhered to surrounding organs. Sometimes the hepatic tissue underneath it may appear unaltered, though it is compressed, the bulk of the entire organ shrunken, and there is usually extensive destruction of the lobules. Sometimes the fibrous growth occurs most extensively in the interior of the organ, its prolongations along the portal canals being principally involved. Perihepatitis is common in syphilitic cirrhosis, the capsule being thickened and adherent to surrounding organs, while fibrous bands pass into the substance of the liver, undergoing contraction, causing deformity and resulting in fibrous scars. These scars represent the sites of gummatous deposits, these being most numerous along the attachment of the suspensory ligament.

In *fatty cirrhosis* the size of the liver is permanently increased, the surface presenting a smooth or slightly granular condition with yellowish-white or anæmic hue, the general appearance being that of a fatty liver, though when cut its resistance to the knife determines the difference, evincing the presence of a large amount of fibrous tissue. The excessive amount of fat deposited in the lobules accounts for its bulky appearance, though this is not a distinctive feature as fatty degeneration occurs in the peripheral zone of the lobules in all forms of cirrhosis.

Hypertrophic cirrhosis proper, or biliary cirrhosis, is characterized by the retention of bile in the small biliary passages and their radicles, with infiltration of pigment into the lobules and connective tistue. Fibrous deposits in the lobular capillaries impart an early firmness which resists contraction and results in permanent enlargement, and pigmentation imparts a deep-brown or black color to the affected tissues. The obstruction to the portal circulation gives rise to numerous pathological conditions about the tissues drained by its radicles. Without an extensive collateral circulation speedily fatal results must soon follow. In spite of this, engorgement of the gastro-intestinal radicles results in catarrh of the stomach and bowels, exudation of blood into the alimentary canal signalized by hæmatemesis and melæna, and effusion of serum into the peritoneal cavity (ascites).

Such changes, however, are modified by collateral venous connection between the general and portal venous systems, this sometimes being very extensive and at other times very restricted. The hemorrhoidal plexus communicates with radicles of the portal system and of the internal iliac, through which the surcharged portal vessels may find some relief. The left renal vein anastomoses with the radicles of the duodenum and colon. The phrenic vein may anastomose with superficial branches of the portal vein. New channels are sometimes formed within the liver, between the portal and hepatic veins. Adhesions which may form between the liver and other organs may develop branches of sufficient size to convey considerable of the obstructed blood. A venæ comites of the obliterated umbilical vein may become dilated and accompany the round ligament to the umbilicus, and this may anastomose with the internal mammary and epigastric veins. When this anastomosis is well marked, a circle of dilated veins (caput Medusæ) is to be observed around the umbilicus. Other branches accompany the suspensory ligament and become enlarged, anastomosing with the diaphragmatic veins, and thus joining the azygous veins. The œsophageal and gastric venous radicles also anastomose, thus affording an additional channel for the escape of the obstructed portal circulation.

Symptoms.—The extent of the collateral circulation will determine, to considerable degree, the severity of the symptoms. When this is extensive, a subject of hepatic cirrhosis may not suffer marked disturbance, and may continue in comparative comfort for a long time. However, this is not usually the case. Hepatic congestion is soon announced by sensation of fullness and weight in the right hypochondrium with dypsnœa, from crowding beneath the diaphragm; the hepatic region is protuberant, tender upon pressure, and the area of dullness about the liver is increased, while palpation may detect the hard edge of the organ a hand's breadth below the ribs. General malaise, with headache, anorexia, nausea, furred tongue, and disgust for meats, soon attends. Gastric irritation now develops, the patient being attacked with retching and empty vomiting, especially in the morning upon waking from slumber, varying attacks of diarrhœa being interspersed. The digestion of food is soon attended by all the distress of gastric catarrh, and the tongue may become red and pointed. The countenance assumes a muddy, icteric hue (though not a markedly jaundiced one), the skin becomes dry and harsh, the spleen enlarged, hemorrhoids appear, and a caput Medusæ, with enlargement of the superficial abdominal veins, may be observed. Emaciation and cachexia rapidly encroach, and the patient may be subject to vertigo, prostration, and occasional pangs of acute pain in the right hypochondrium due to intercurrent attacks of perihepatitis.

As the disease continues, hæmatemesis and melæna occasionally occur, and the gastric symptoms become still more aggravated. Palpation of the right hypochondriac region may now enable one to detect a lessening in the size of the liver, a sense of hardness and irregularity of the surface being imparted to the touch. Tympanites of the abdomen appears, and ascites follows at a later stage, while œdema of the feet and ankles, and, finally, general anasarca, may be developed. Jaundice is not a marked symptom except in biliary cirrhosis, as, though there is obstruction to the secretion of bile, the ducts remain open for its discharge into the intestine. The mind usually remains clear to the last, though delirium or coma may attend the closing scene.

The symptoms of *biliary cirrhosis* are distinctive. The hypochondric fullness is marked, and jaundice is an early feature. Œdema of the face and limbs, profuse sweats, hemorrhages, with increasing jaundice, though without marked emaciation, attend. Ascites is not so common as in the atrophic forms, and the hepatic enlargement is progressive, the enlarged liver being smooth and rounded. Enlargement of the spleen is noticeable. After a time the disease is likely to terminate with symptoms of acute febrile jaundice, a

chill ushering in febrile symptoms, with delirium, coma, convulsions, and death. The disease may exist in the chronic form for two years or more, the acute termination lasting ten days or two weeks.

Diagnosis.—The former habits of the patient will aid in a diagnosis. An individual addicted to alcohol who suffers with hepatic enlargement, gastric disturbance, hemorrhages from the stomach and bowels and ascites, is probably a subject of cirrhosis. The firm, hard or nodulated liver, felt upon palpation during the second stage, with evident contraction of bulk, is a strong suggestion of the disease. Palpation should here be made after paracentesis, in order to examine the organ carefully. Enlargement of the spleen existing coincidentally, is additional evidence of cirrhosis. A history of syphilis with the preceding developments may be considered confirmatory of a suspicion of cirrhosis, and young children with syphilitic antecedents are liable to it. Cancer of the liver will be differentiated by the marked cancer cachexia and rapid loss of strength, which is more evident than in cirrhosis. Obstruction of the portal vein by fibrous thrombosis may be difficult to distinguish from cirrhosis, as the symptoms are very similar. The enlargement which attends the early stage may be difficult to distinguish from fatty liver, though there is absence of pain and gastric complication.

Prognosis.—The prognosis is usually unfavorable, though where there is extraordinary collateral circulation the subject may survive for years. However, hepatic cirrhosis terminates fatally within a few years in most cases, and it may run its course in a few months. After ascites and hematemesis appear there is little hope of a favorable termination, though if the disease be diagnosed early and proper treatment be employed there is considerable probability of improvement.

Treatment.—All exciting causes should be avoided. Alcoholic liquors must not be allowed under any circumstances, and stimulating and highly seasoned food should be forbidden. The most bland and unirritating food should be chosen, a milk diet with crackers and stale bread being preferable. To relieve gastric disturbances, plenty of hot water should be taken, aërated waters being beneficial. A careful study of some reliable work on diet will be useful to the patient, that he may possess an intelligent idea of what is best suited to his case. Sometimes milk causes "biliousness," and other food may be required, buttermilk, koumiss, whey, or malted milk being preferable. After ascites has appeared fluids had better be dispensed with, as they tend to increase the amount of dropsical accumulation. A dry diet, consisting of stale bread with a small quantity of meat, such as the white flesh of fish, oysters, sweetbread,

liver, roast or broiled beef, and eggs, may be consumed, in limited quantities. Tea and coffee should be avoided. Certain vegetables, such as radishes, onions, garlic, etc., stimulate the liver; though others, as spinach, asparagus, tomato, squash, pumpkin, celery, lettuce, oyster plant, etc., are allowable. Fruits are commendable to encourage normal evacuation, though they should be selected with a view of avoiding a stimulating influence upon the liver. Strawberries, grapes, oranges, apples, and peaches may be eaten. The same precaution should be taken as in gastric catarrh; the food should be taken slowly and thoroughly masticated, and its amount should be limited to only enough to supply the needs of the body, it being better for the meals to be taken alone.

When treatment is begun at an early stage of the disease, while the liver is hyperæmic, much benefit will follow the use of *potassium chloride* 3x, five grains in half a glass of water, dose, a teaspoonful every two hours, while awake. This will effectually prevent the organization of the newly-formed plastic material, and tend to permanent recovery. After the advanced stage has been reached, medication is of little use except to temporarily relieve aggravated symptoms.

Gastric fermentation may be temporarily relieved by lavage or by the use of hydrozone, and nausea and vomiting may be treated with minute doses of aconite and rhus tox. When ascites becomes so extreme as to cause much discomfort, paracentesis abdominalis should be resorted to, and this should be repeated sufficiently often to prevent unpleasant crowding of the abdominal cavity. Chionanthus and chelidonium may afford some benefit in biliary cirrhosis. Syphilitic cirrhosis should be treated with iodide of potassium. Protonuclein may be found of service here, administered early.

Abscess of the Liver.

Synonym.—Suppurative Hepatitis.

Etiology.—Abscess of the liver occurs as a single, large accumulation of pus in one lobe of the organ, or as numerous small, separate accumulations of purulent material, scattered throughout the liver-tissue.

Large or single abscess of the liver occurs most frequently in tropical regions, either idiopathically or as a sequel to dysentery, and the fact that the amœba coli is invariably found in the pus in such cases points strongly to its influence as an etiological factor. It is a common disease in India, especially among Europeans who indulge freely in the use of alcoholic drinks, and is not rare in the southern states of our own country. Large single abscesses of the

liver may result from traumatism, blows received about the middle of the body and falling upon the organ frequently result in suppuration. Pugilists and railroad brakemen are most liable to suffer from this form, the crushing effect of injuries received while coupling cars rendering the vocation of brakeman especially hazardous in this respect. It is extremely rare among women, not more than four or five per cent. of all cases occurring in females.

Multiple abscess of the liver is usually due to dissemination of purulent material along the portal canals from infection of the portal blood outside of the liver, though the suppuration is sometimes due to irritation of the bile-passages from gall-stones (suppurative cholangitis); parasites in the liver, such as the echinococcus, intestinal worms, or the fluke-worm; tuberculosis of the liver; or penetration of its substance by such foreign body as a needle or fish-bone, which has perforated the œsophagus and wandered into the hepatic tissues.

Embolic or *pyœmic* abscesses of the liver may develop from general pyæmia, the infection entering through the hepatic artery; or it may arise from causes originating among the radicles of the portal vein or in the vein itself, when suppurative thrombosis of that vessel occurs. The ulceration of typhoid fever may afford a nidus from which infective material may enter the portal vein and reach the liver. Dysentery, rectal disease, appendicitis, or pelvic abscess may furnish the element of multiple suppuration of the liver, the ramifications of the portal vein distributing it throughout the organ. In new-born children the infection may enter through the umbilicus.

Pathology.—In single abscess of the liver the right lobe is the usual seat of suppuration, an extensive area of destruction being involved. Sometimes several quarts of pus may be discharged from an abscess of this character at one time. Instead of a single abscess, two or more large ones may exist, these either remaining single or, as the disease progresses, coalescing. The pus may be limited by an abscess-wall or not, there frequently being no limiting membrane, the confines being irregular projections of semi-disorganized liver-tissue, projecting into the abscess-cavity. The purulent material varies according to the origin of the disease, that of tropical abscess being of a reddish-brown color, resembling anchovy sauce, possessing a peculiar odor resembling that of chyme, and containing amœbæ coli in great numbers. In traumatic cases, the pus may be flocculent and thin, or thick, creamy, and yellowish-green or brick-red in color, from the staining of bile or bilirubin. In traumatic abscess the pus is sterile of microörganisms.

As the disease continues, the pus gradually works its way toward the surface of the liver and finally perforates the limiting structures,

sometimes opening into the pleural cavity, sometimes perforating the diaphragm and discharging through the lung, sometimes penetrating the pericardium, vena cava, intestine, stomach, kidney, gall-bladder, or peritoneal sac. Adhesion of the peritoneal surfaces may occur, and the pus burrow its way to the surface through the abdominal or thoracic wall. Nine per cent of all cases discharge through the right lung, and five per cent empty into the right pleura. With modern surgical knowledge, spontaneous discharge of pus will be anticipated, in these cases, by early evacuation.

Multiple abscesses arising from pylephlebitis are distributed to the ramifications of the portal vein, the abscesses usually lying near the capsule. Numerous white points frequently appear beneath the capsule, marking the locations of purulent deposits, and if these be traced by probing they are found to communicate with the portal veins. The liver, especially the right lobe, is markedly enlarged, the organ rising into the thorax and extending as much as a hand's breadth below the margin of the ribs. The suppuration may extend along the branches of the portal vein, even into the main branch, and thrombi may be distributed to its branches, in various parts of the liver. In obstruction by gall-stones, the biliary ducts and gall-bladder may be filled with purulent material. The pus is fœtid, greenish-yellow, and it may be flocculent, though it is frequently thick and laudable. Large abscesses may form about hydatid cysts, the presence of echinococci indicating their morbid character.

Symptoms.—In rare cases, tropical abscess may arise insidiously and rupture without warning, sudden death occurring from an unsuspected cause. Usually, however, the onset of the disease will be announced by chills, following a short period of malaise, during which the temperature may be subnormal. The chills may occur periodically, and be followed by paroxysms of fever which decline by sweating, the disease resembling an attack of ague, the tendency to perspiration being marked, especially while the patient is sleeping. Sometimes there is but the one chill, the succeeding fever being remittent, and rising in the afternoon. In other cases, there may be no febrile action, this occurring most commonly in chronic cases. The temperature rises, in febrile cases, as high as 103° or 104° F. during the afternoon, declining toward morning. Pain, fullness, weight, and tenderness in the right hypochondrium attend these symptoms, the area of liver-dullness in the right thorax being enlarged, and the liver extending downward into the abdomen a hand's breadth below the margin of the ribs. Fluctuation may occasionally be detected upon palpation. Respiration is impeded by the encroachment upon the thoracic space, and full inspirations are attended by increase of pain in the right hypochondrium. The pain

varies in character, to correspond with the location of the abscess, it being dull and aching when deeply seated, and sharp and lancinating when so near the surface as to affect the peritoneal covering. Pain at the point of the shoulder or angle of the scapula is a frequent symptom. Sometimes diffuse peritonitis may arise, and the lancinating pain become severe and wide-spread.

Gastric irritation may attend, the tongue being heavily loaded; and the countenance is dull and expressionless and presents a muddy, icteric hue. Typhoid symptoms appear early; and delirium, typhomania, coma, or convulsions may soon develop.

When the purulent accumulation points toward the thorax, characteristic symptoms arise. If the lung be perforated, a violent and harassing cough arises, and soon the purulent discharge is expectorated, the reddish, characteristic material resembling anchovy sauce, denoting the condition in tropical abscess. When the thorax is invaded within the pleural cavity, symptoms of pyothorax arise.

In multiple abscess of the liver the symptoms may not be so active, pyæmic symptoms attending, the liver presenting enlargement, and the skin assuming an icteroid hue. The pain and tenderness may not be so acute as in single abscess, though there is appreciable enlargement. The disease runs a more chronic course, but is more likely to prove fatal from septicæmic complication.

Diagnosis.—The chills, febrile symptoms, and enlargement of the liver, with pain and tenderness, will usually call attention to the liver as the seat of inflammatory disease. Aspiration will decide as to whether there is an accumulation of pus. It is to be recollected that hepatic abscesses involve the upper portion of the organ in most instances, and here is the place to aspirate for pus, several trials being sometimes necessary in multiple abscess in order to intersect one, and locate it with the point of the needle.

Prognosis.—The prognosis of pyæmic abscess of the liver is invariably unfavorable. Single abscess, under modern surgical methods, may often be brought to a favorable termination. If the disease be diagnosed early, and potassium chloride 3x be administered early and perseveringly, arrest of suppurative action may be accomplished in many cases of traumatic, if not in tropical abscess.

Treatment.—The early administration of *potassium chloride* 3x, five grains in half a glass of water, dose, a teaspoonful every hour, is the proper medication for the early stages. If expectoration of pus suggest the discharge of the abscess through the lung, a conservative course may be better than a radical one, the complications being met as they arise and aspiration of the lung postponed. The general treatment of septicæmia will be applicable after there has been purulent accumulation.

Section of the abdominal muscles, down to the liver, with subsequent stitching of the liver-tissue to the abdominal wall, so that a surface is left for opening, and free drainage of the abscess, is probably the most successful method of treating this disease. The open abscess then becomes an ulcer which is easily treated until complete recovery results.

The *diet* should be free from fats and liquors, and consist of small quantities of fluid, predigested food, such as pancreatinized milk and malted milk, and animal broths free from fat. If there does not seem to be enough nourishment in such diet, eggs, beaten in milk, may be used sparingly, food being taken in small quantities at a time, every three hours.

New Growths in the Liver.

The principal new growths which occur in the liver are carcinomata, sarcomata, angiomata, and adenomata. To these may be added cystic accumulations, which, though not strictly new growths, are usually considered in this relation.

Carcinoma of the liver may occur as a primary affection, and secondarily from infection by continuity of other organs, through the lymphatics, and, at an advanced stage of the disorder, through the portal vein or hepatic artery. Women are more liable to it than men, and it is rare before the middle of life, tendency to it increasing after that time to the sixty-fifth year.

Pathology.—Cancerous growth occurring near the entrance of the portal vein may so obstruct the circulation as to result in ascites and dilatation of the radicles of its collateral branches. In some cases the cancer-growth may be localized in a small portion of the liver as a primary growth, and numerous nodules, of later development, arise secondarily. In other instances, the entire organ may be involved in primary cancerous growth (massive cancer). Infiltration of the liver with cancerous elements is common in secondary infection from the lymphatics or portal vein, the development of fibrous growths about the cancer deposits causing a general resemblance to cirrhosis. The shape of the liver may be variously altered; one large growth may be surrounded by numerous smaller distributed nodules, or the entire surface may be covered with with small, irregular nodules, sometimes projecting from the surface, at others imperceptibly grading into the general surface, and, in other instances, presenting crater-like depressions. Hemorrhages may occur into the nodules, and suppuration occasionally, though rarely, ensues. Pressure upon the biliary passages, from contraction or nodular crowding, is not an uncommon condition, retention of bile

and hepatogenous jaundice resulting. In secondary cancer of the liver, the organ is often enormously enlarged, its weight sometimes exceeding twenty pounds. The nodules project beneath the capsule, and may be plainly felt through the attenuated abdominal walls. Various degenerative changes may occur in the morbid structures, such as hyaline or fatty degeneration, or sclerosis. When the disease originates in the bile-passages, it is usually associated with irritation from gall-stones, and the cancerous growth frequently arises in the base of the gall-bladder. Biliary obstruction from extension of the process to the common or hepatic duct is liable to arise, with retention of bile. In some cases the disease arises primarily in the ducts. Localized peritonitis often gives rise to adhesions of the capsule to surrounding peritoneal surfaces.

Symptoms.—The symptoms of hepatic cancer may be obscure in the beginning, pain, fullness, and icterus being attendants of various other hepatic disturbances. The cancer cachexia, however, becomes marked before long, and gastric symptoms attend, as well as those of biliary and portal obstruction. A knowledge of primary cancer preëxisting in some other part, such as the rectum, uterus, pylorus, or mamma, renders the diagnosis of secondary cancer of the liver easy, but primary cancer of the organ requires more care. When the cancerous growth is located near the periphery of the liver, the nodulated surface, with its crater-like depressions, may be palpated through the attenuated abdominal walls, and this, in connection with the loss of strength and flesh, cachectic pallor of countenance, and tendency to rejection of food by the stomach, with pain in the right hypochondrium or beneath the right shoulder or in the dorsal region opposite, suggests the condition strongly. Ascites usually arises as a result of obstruction to the portal circulation, and œdema of the feet appears at a late stage.

Though hepatic cancer may run its course without pain, jaundice, or ascites, these are usually prominent symptoms. The pain is often lancinating, this being due to involvement of the peritoneal surface. The temperature is normal or subnormal and the pulse small and rapid.

Enlargement of the cervical and inguinal lymphatics is often present from cancerous infiltration, and there are hematemesis and melæna, and hemorrhages from the mouth and vagina. Ecchymotic spots may appear on the skin.

Diagnosis.—The enlarged, irregular-shaped liver suggests the condition, and umbilication of the nodules establishes the diagnosis without question. When palpation fails to distinguish this, the cancerous cachexia, in connection with the local disturbances, such as pain, irregular enlargement of the liver, and persistent gastric dis-

turbance, can hardly be mistaken. Aspiration will exclude abscess from the diagnosis.

Prognosis.—The prognosis of hepatic cancer is invariably unfavorable. The pain and other discomfort, however, may be modified by rational treatment, and much suffering thus prevented. Medullary cancer of the liver is a rapidly fatal disease, usually terminating within from two weeks to four months. The average duration of cancer of the liver is one year.

Treatment.—If treatment is begun early, and properly persevered in, many of the severe symptoms may be averted, and the progress of the disease stayed considerably. *Echinacea* not only relieves the pain of cancer, but it seems to fortify the system against the rapid inroads of the disease, retard the rapidity of cancerous growth, improve assimilation, and prevent the rapid dissemination of the cancer-elements, while it lessens anæmia and cachexia A steady use of this remedy is therefore advisable, ten or fifteen drops of the saturated tincture of the recent plant being administered every four hours. *Chelidonium* may be alternated with this, five or ten drops of the specific medicine being administered every four hours. Besides a supposedly antidotal influence against cancer, this remedy encourages normal hepatic processes, and assists in the elimination of bile from the biliary passages, thus guarding, in some degree, against hepatogenous jaundice. The diet should be nutritious and digestible.

Sarcoma of the liver is a rare disease, though it occurs in a few cases primarily, and somewhat more frequently secondarily. It may occur in the form of lympho-sarcoma, myxo-sarcoma, or glio-sarcoma. Melano-sarcoma is a variety in which the morbid tissues are pigmented, presenting the appearance of dark granite or deep black, mottled with streaks of white like dark marble; and this is the form usually prevailing. It occurs as a secondary affection following sarcoma of the eye or skin. The liver becomes much enlarged, and is either uniformly infiltrated with the morbid growth or nodular masses may be distributed through its structure, the surface, however, presenting a uniformly smooth appearance. As the blood distributes sarcoma readily, numerous metastases are liable to attend the liver-affection early, many other organs being involved. The disease is most liable to affect the young and those before middle life.

Adenomata of the liver occur occasionally, appearing in the form of small encapsulated tumors, having the structure of the liver. Their presence is, however, rare.

Angiomata of the liver is most liable to occur in children, and consist of masses of dilated blood-vessels about the size of a walnut,

of dark reddish color. They sometimes attain a much larger size, increasing the bulk of the liver, though the liver-tissue is not altered. They usually occur singly.

Cysts of the liver may be single or multiple, and usually occur in connection with congenital cystic kidneys. Hydatid cysts (from echinococci) will be considered in another place.

The *diagnosis* of sarcoma is often difficult, the pain and enlargement being common to other morbid conditions of the liver, though accompanying sarcoma of the skin or eye will furnish a valuable suggestion. *Angiomata* and *adenomata* are recognized with difficulty, though an adenomatous condition generally would lend color to a suspicion of adenomatous growths in the liver if hepatic disturbance attended. Persistent hepatogenous jaundice, due to pressure upon the biliary ducts, gastric disturbance, and enlargement of the liver, are among the constant symptoms of all new growths of the liver.

The *treatment* cannot be specified. Such urgent symptoms as may arise should be met by the judicious administration of what, according to our knowledge of materia medica, seemed to be most urgently demanded, care being taken to avoid opiates, so far as possible. If angioma be diagnosed, calcium fluoride 3x may be administered with some hope of benefit.

Fatty Liver.

Etiology and Pathology.—Fatty liver, in some form, is of frequent occurrence. It may occur as an infiltration or a degeneration under abnormal conditions, and physiological infiltration of the cells is constantly present during health, and increased after the ingestion of fatty food. The liver-cells which lie near the branches of the portal vein, i. e., the circumferential cells of the lobules, receive the fatty deposit first, and, in abnormal infiltration, here is where the process is most observable. For the disposal of fat in the portal blood, active oxidation must occur before it can be received by the hepatic cells, and to lack of proper balance between fat-supply and oxidation is referable abnormal infiltration of the liver with this material. (1) An excess of non-nitrogenous material in the blood may defeat the efforts of normal oxidation, and an undue amount of fat thus be left to be taken into the hepatic cells, this condition accompanying excessive deposit of fat in other parts of the body and constituting general obesity, a condition not necessarily attended by impairment of function or danger to hepatic integrity. (2) A normal or even small amount of fatty material in the portal circulation may result in fatty infiltration when there is lack of a proper supply of oxygen for its consumption; and fatty infiltration

of the liver is not rare in phthisis, anæmia, and other cachexiæ, the deposition of fat then occurring in the liver while other parts of the body are undergoing emaciation.

Fatty degeneration or fatty metamorphosis implies a destruction of the individuality of the hepatic cells and the occupation of their sites with fat-globules and débris of the preëxisting cells. This is a much more serious condition; for, when general, it implies cessation of hepatic function. It is due to inpairment of the vitality of the hepatic cells, through the local influence of irritating or poisonous substances, such as toxines and phosphorus, and from lack of nutrition, as when the normal blood-supply to a lobule or group of lobules is impoverished or cut off, as in anæmia, cirrhosis, etc.

The fatty liver is uniformly increased in size, and its surface is smooth and presents a bloodless, pale appearance. On section, it is dry, and it cuts as though greasy, and leaves a fatty coating on the knife. Sections, and even the entire organ, though greatly enlarged and increased in weight, may float in water.

Symptoms.—The symptoms are not very striking or definite. Enlargement in the right hypochondrium is present, the edge of the enlarged organ sometimes being distinguishable by palpation below the navel, soft and doughy on pressure. It is not, however, painful or sensitive. Gastro-intestinal symptoms, referable to pressure and portal obstruction, are frequently prominent; for, though there may not be sufficient pressure upon the portal canals to originate ascites or splenic enlargement, there is enough to congest the gastro-intestinal mucous membrane and interfere with the digestive functions. There may be vomiting, with gastric catarrh, attacks of diarrhœa, and other symptoms of gastro-intestinal dyspepsia, with loss of strength, anæmia, drowsiness, and despondency. Dyspnœa arises, both from debility, and pressure upon the diaphragm. The skin has a peculiar smooth, "velvety" feel, and the tissues are flabby and inelastic. The biliary functions are not interfered with unless there is extensive destruction from metamorphosis of the hepatic cells, and jaundice is seldom noticeable. . When metamorphosis of the hepatic cells is extensive, rapid anæmia, exhaustion, delirium, and collapse are liable to occur at any time, the stools presenting a pale, clay-colored appearance, attacks of diarrhœa then frequently occurring.

Diagnosis.—The large, rounded liver, with smooth surface and inelastic tissues, will not be confounded with cirrhosis or cancer, in which nodular or granulated projections are distinguishable. The only disease liable to be confounded with it is waxy liver, and here the skin is dry and pale, while in fatty liver, it is soft, velvety, moist and shining. In fatty liver the blood is hydræmic, while in

waxy liver it is leukæmic. The history of waxy liver may also assist in determining the matter, as syphilis is liable to result in waxy liver, while fatty liver seldom follows it. The waxy liver is hard and firm, while the fatty liver is soft and doughy. In fatty liver the urine is normal, while in waxy liver it is albuminous, and often contains casts. The spleen is enlarged in waxy liver, but remains normal in fatty liver.

Prognosis.—The character of the fatty accumulation will determine its gravity. Fatty infiltration is not a serious condition, but fatty metamorphosis, when extensive, is of serious nature.

Treatment.—An abstemious diet and active occupation are conducive to recovery in all curable cases. Plenty of out-door exercise in elevated regions, under sanitary conditions, is an imperative part of treatment. *Oxygen gas*, either by inhalation or by rectum, improves the power of the portal blood to dispose of fatty material, and is always to be commended. Phthisis, syphilis, and other conditions predisposing will demand special treatment. Sugar, starch, fats, malt liquors and alcoholic drinks should be considered as pernicious, as a rule, though fatty liver may exist in anæmic or prostrated conditions where a judicious use of some of these substances may be required. *Berberis aquifolium* may be employed to improve the digestive power, and gastric catarrh may require hydrozone or lavage to prevent fermentative action.

Amyloid Liver.

Synonym.—Waxy Liver.

Etiology.—The principal cause of waxy liver is syphilis. It occurs most frequently in males between twenty-five and fifty years of age. Prolonged suppuration, and chronic diseases of bone, are other prominent causes, phthisis, ulceration of the bowels from chronic dysentery, chronic pyelitis, and rickets, being most frequently predisposing causes after syphilis. Prolonged convalescence from infectious diseases, especially malaria, and any form of cachexia attended by wasting and impoverishment of the blood may finally develop amyloid degeneration of the liver.

Pathology.—The amyloid change begins in the radicles midway between the center and periphery of the lobules, and extends to the minute branches of the hepatic artery. A material of nitrogenous, homogenous, translucent appearance, with dull, glistening surface infiltrates the walls of the capillaries in the median zone of the lobules, and extends to the interlobular vessels and connective-tissue, the cells being but little involved. When the affected tissues are stained with iodine, the morbid deposit assumes a rich mahog-

any-brown. The entire liver is enlarged, firm and resistant, the edges sharply defined and the surface smooth.

Symptoms.—Enlargement of the liver, causing bulging in the right hypochondrium, with increased area of liver-dullness, without pain or tenderness, is the principal symptom. There is no biliary obstruction, and jaundice is absent, though the stools may be light-colored. The spleen is occasionally involved, its bulk being augmented.

Diagnosis.—This is usually easy, as the history of the case will account for the gradual and progressive enlargement of the liver without pain or tenderness. Long-standing suppuration, syphilitic antecedents or persistent cachexia, followed by such symptoms, will naturally support a theory of amyloid degeneration.

Prognosis.—Unfavorable. Syphilitic cases may be modified and life prolonged by proper treatment, but the inevitable result will finally be death. The disease runs a slow course, and may drag along for months, and sometimes years, such complications as diarrhœa, purulent peritonitis, perihepatitis, fatty or waxy kidney, pulmonary œdema, pulmonary gangrene, etc., appearing meanwhile. Death may finally result from exhaustion, anasarca, diarrhœa, uræmia, or other causes arising from varying complications.

Treatment.—Syphilis should be properly treated, and causes of prolonged suppuration removed if possible. Primary diseases should be rectified when practicable, and anæmia and cachexia corrected by all available measures. Each case will suggest its medicinal treatment, it being recollected that no specific remedy can be recommended for amyloid degeneration. All that can be done is to strive to correct the dyscrasia upon which the morbid change depends.

The diet should be considered, and sugars and starch avoided, and, when the stomach is fairly active, lean beef should be the principal diet. When digestion is greatly impaired, predigested foods may be required, beef peptonoids and pancreatinized milk being representative forms of diet. When the disease has advanced sufficiently far to be readily diagnosed, little time will usually be left for treatment, a fatal termination soon attending.

Tuberculosis of the Liver.

Tubercles may be deposited in the liver during the course of general tuberculosis, and in connection with tubercular disease of the intestines and mesenteric glands, though there are few distinctive features attending, and they attract little more than pathological interest. In miliary tuberculosis, they are distributed throughout

the liver-tissue in small masses, while in the chronic forms they may occur as a few large tubercular deposits. In chronic tuberculosis of the liver there is usually considerable increase in the amount of connective tissue, and the deposits are liable to be associated with chronic perihepatitis or peritonitis. On account of the extensive proliferation of fibrous-tissue atttending, chronic tuberculosis of the liver is usually designated as "tubercular cirrhosis."

VIII. DISEASES OF THE BILE PASSAGES.

Catarrhal Inflammation of the Biliary Passages.

Synonym.—Catarrhal Jaundice.

Etiology and Pathology.—Reference to this disease has been made under jaundice, but completeness demands notice of it here. It arises from congestion of the mucous membrane of the common bile duct in most cases, though it has been asserted that the inflammation may begin in the smaller passages and extend to the larger canal. Duodenal catarrh, accompanied by indigestion, usually originates it, the inflammatory action extending inward, from the intestinal mucous membrane. Obstruction of the biliary passage may be due to accumulated and inspissated mucus in the passages, or in the common duct alone, the common point of obstruction being in the *pars intestinalis*, that portion which extends into the intestine. It occurs frequently in young persons, though all ages are liable to it, indiscretions in diet, colds, malarial attacks, and all causes which predispose to portal obstruction, being liable to be followed by it. It sometimes occurs in fevers, pneumonia, etc. Emotional disturbances are sometimes followed by jaundice supposed to be due to this condition, and it may occur *epidemically*, from unknown causes.

Symptoms.—Where the disease is due to continuity of intestinal irritation, it is apt to be preceded or attended by indigestion, flatulence, and constipation. The skin rapidly assumes an icteric hue, the color being bright yellow, and never the greenish tint observed in some cases of grave or long-continued jaundice. Slight fever may attend, though the temperature is rarely above 101° or 102° F., and it may not be elevated at all. In the epidemic form, however, there is liable to be an initiatory chill, followed by fever and headache. The bowels are constipated and the stools are clay-colored, while the urine contains bile pigment. Though the pulse may be quickened in the epidemic variety, and may sometimes be normal in other cases, it is usually abnormally slow, falling as low as forty, thirty, or even twenty beats per minute. Slight enlarge-

ment of the liver may occur, though its size may be normal or considerably enlarged. The duration of the disease varies from two to twelve weeks, the first indication of returning health being a return of the normal color of the stools. General malaise, muscular pains, nausea and anorexia, dizziness, drowsiness, and indisposition to exercise, are frequently present.

Diagnosis.—The symptoms are of such a mild nature and occur so suddenly, while the patient is about, or follows suddenly upon an acute attack of gastro-intestinal irritation, that there is little danger of a mistaken diagnosis. Other forms of jaundice come on gradually, or are much more severe in their symptoms. In jaundice from organic disease of the liver, there is emaciation, ascites, and other indications of portal obstruction. Weil's disease and malignant jaundice present characteristic symptoms.

Prognosis.—The prognosis is favorable, rational treatment usually bringing the disease to a speedy close. Even badly treated cases may recover after a time, without serious complication or sequelæ.

Treatment.—The treatment of this form of inflammation will depend upon the conditions presented. If febrile symptoms appear, *aconite* and *rhus tox.*, or other appropriate sedative treatment may be employed in the beginning. Malarial manifestations must be appropriately met, periodicity being interrupted with *quinine* and this may be followed by ten-drop doses of *grindelia squarrosa* (green plant tincture), repeated every four hours during the day and evening. The most appropriate remedies for the biliary obstruction are *chelidonium* and *chionanthus*, either in combination or singly. *Enemata* are useful to assist the action of the chologagues, and the use of the salt water galvanic *electrode* in the lower bowel, with the positive pole applied over the hypochondriac region, is an excellent aid, and even curative measure, in a large majority of cases in which it is tried.

Gall-Stones.

Synonyms.—Cholelithiasis; Biliary Calculi.

Definition.—Concretions which form in the gall-bladder, due to inspissation or concentration of the bile, from long retention.

Etiology.—It is believed that defect in the sodium salts favors the precipitation of cholesterin, of which the concretions largely consist. Inactivity of a person tends to the production of biliary calculi, those of sedentary habits being most prone to them, the majority of cases (75 per cent) occurring in women. Pressure upon the cystic duct doubtless favors their formation by obstructing the free flow of bile, lacing and pregnancy thus rendering women excep-

tionally prone to the disease, about 90 per cent of the cases occurring in women affecting those who have borne children. It has been asserted that twenty-five per cent of all women past sixty-five years of age are subject to gall-stones. The majority occur after middle-life, the disease being rare in persons less than thirty-five years of age. A fatty diet, an excess of animal food, and alcoholic drinks are supposed to figure as causal factors.

Pathology.—The number of gall-stones in the cyst and biliary passages may vary from one to a thousand. Where there are great numbers, they may be very small—not larger than a small bird-shot. Where there is but one it may be very large, sometimes the size of a lemon, and one five inches in length has been reported. The very small concretions may form in the small bile-ducts, but the large ones originate in the gall-bladder. When there are numbers of these concretions in the gall-bladder they are marked with facets, due to pressure or friction from one another being polygonal in form. When there is but one, or a few which are not crowded, they may be oval or globular in shape. If a smooth section of a gall-stone be made through its center, it will be seen to consist of concentric layers, surrounding a nucleus, which may consist of bile-pigment, a cast of an hepatic duct, crystals of cholestrein, cholate of lime, a blood-clot, a fluke-worm or other parasite, etc. The separating lines between the layers or concentric rings may be crossed by crystalline radiations of cholesterin; however, this substance constitutes about eighty per cent of all gall-stones. In some cases there may be no radiation, the concentric layers being distinctly separate. The external crust varies in character and consistency, though the internal structure is composed largely of cholesterin. Sometimes it may be composed principally of carbonate of lime, and will then be rough and of whitish color, while in other cases it may consist of a mixture of cholesterin and pigment, the color being of a greenish-yellow or brownish color, and smooth. It is seldom that they undergo erosion or disintegration, their structure remaining permanent, unless they increase in size by the addition of material to their surfaces. The gall-bladder resists the local influence of the concretions for a long time, and may not be very much altered by their presence, though the mucous membrane may finally become catarrhal and eroded, and finally the entire wall may become thickened and fibrous, the cyst being contracted and hardened with fibrous deposits, or converted by calcareous degeneration into an unyielding, stony mass. When the gall-bladder is impacted with calculi its walls may be ulcerated, and perforation may occur with escape of the calculi through the abdominal wall.

Various peculiar changes follow the impaction of a calculus in

the cystic duct. Ulceration and perforation of the duct with escape of the calculus into the abdominal cavity may attend. Dropsy of the gall-bladder is among the possibilities of the case, the cyst becoming enormously distended with a thin mucoid fluid, several pints being sometimes pent up in its cavity, giving rise to a circumscribed tumor, which may be mistaken for an ovarian cyst. When the calculus passes through the cystic duct, its motion is rotary, from the peculiar spiral arrangement of the mucous folds of this passage. When impaction of the cystic duct occurs, symptoms of jaundice may be entirely absent, while impaction of the common bile-duct would be attended by pronounced hepatogenous jaundice, the bile being retained and absorbed into the circulation. Ulceration and perforation of this duct may result from a retained and impacted gall-stone, as well as in the case of the cystic duct. From such a source may arise fistulæ opening through the abdominal walls, into the duodenum, colon, stomach, ureter, pleural cavity, vena cava, vagina, and other organs, leading from the point of exit of the calculus through the wall of the gall-bladder, cystic duct, ductus communis choledochus, etc.

Symptoms.—The most common symptom of gall-stone is *biliary colic*, the occasional passage of a calculus being attended by excruciating pain, of paroxysmal character, during its course along the bile-ducts to the intestine. The first experience of this kind usually marks the establishment of the "gall-stone habit," the patient being subject to more or less frequent attacks of hepatic colic for months or years, unless proper treatment be instituted to arrest the tendency to their formation. Biliary colic usually arises after some peculiar provoking cause which produces engagement of a calculus in the biliary passage, the predisposing cause probably being pressure from behind, due to accumulation of fluid in the cyst. A full meal, a ride over a rough road attended by jolting, active exercise, etc., being immediately followed by sudden and severe pain in the right hypochondrium, which is aggravated by change of position and pressure, and which radiates to the epigastrium, along the diaphragm, and to the scapulæ, the entire upper portion of the abdomen being sometimes involved. The pain is boring or tearing in character, and comes on in paroxysms, these often being preceded by yawning, rigors, nausea, vomiting, and profuse sweating. The face becomes pallid and clammy, and the patient becomes faint and prostrated; the abdominal muscles are rigid, the pulse is small and oppressed, and the patient rolls about or screams with agony. In a few seconds the extreme pain may subside for a short interval, to return again, this continuing for several hours or a day, when, as the calculus reaches the intestine, the pain suddenly

ceases, and the patient becomes comfortable, though prostrated, and rapidly returns to an ordinary condition of health, with prospects, however, of another attack, within a few weeks, at least. Symptoms of hepatogenous jaundice may attend and follow the attack, the stools being clay-colored, and the skin and conjunctivæ icteric in hue. When the calculi are small and arise in the biliary ducts, the symptoms of jaundice are more prominent than when the single calculus originates in the gall-bladder, and enlargement of the liver is more prominent, distention of the gall-bladder relieving the accumulation when the common bile-duct and cystic duct are involved. Soreness in the right hypochondrium attends and follows the attack for a few hours.

Hydrops vesicæ fellæ, or dropsy of the gall-bladder, arises from chronic obstruction of the cystic duct by gall-stones. The bile is now replaced by a clear, thin, mucoid fluid, which may accumulate in large quantity, the entire abdominal cavity being sometimes filled, the enlargement resembling an ovarian cyst, and sometimes being mistaken for it, adhesion to the broad ligament having been reported. Little if any pain attends, and jaundice is not likely to be present.

Empymœa of the gall-bladder occasionally occurs, a collection of pus accumulating in its cavity, and this is usually associated with gall-stones. An enormous amount of pus may thus accumulate, the quantity sometimes amounting to more than a pint. Final perforation of the cystic walls is liable to occur, with the formation of multiple abscesses in the neighborhood.

Calcification of the gall-bladder sometimes occurs, this usually being a sequel of empyæma. The cystic walls become stony and unyielding, the mucous membrane and sometimes the entire structure becoming infiltrated with lime salts.

Phlegmonous suppuration of the walls of the gall-bladder occurs, though it is rare. The symptoms are of grave character, death soon following hyperpyrexia, intense abdominal pain, rapid prostration and peritonitis.

Atrophy of the gall-bladder is an occasional sequel of irritation from gall-stones. The walls become contracted until the cyst is shrunken to a mere fibrous cord, or a nipple-like protuberance not larger than a pea. Sometimes the cyst is firmly drawn upon a gall-stone.

Diverticula are sometimes formed in the gall-bladder, in which are found biliary concretions.

In chronic obstruction of the *common duct* enlargement of the gall-bladder is not common, a thin, clear mucus being found in the passages. The symptoms of this condition are, paroxysms of chills, fever, and sweating, not unlike those of ague, attended by jaundice,

which deepens in color after each paroxysm, and may continue for months and even years, the paroxysms being attended by severe pain in the hypochondriac region, with gastric irritation and vomiting. Suppurative cholangitis may follow several years of this condition.

Suppurative cholangitis is attended by remittent fever, followed by hepatic abscess, or perforation of the gall-bladder, with abscess between the liver and stomach, with tenderness of the abdomen and septicæmia.

Treatment.—The treatment of biliary colic should be directed to the relief of the severe pain during the paroxysms, and to the prevention of the formation of more concretions in the gall-bladder. If the latter proposition can be carried out, the need of treatment for more serious conditions will probably be done away with.

During the passage of a calculus, a napkin, moistened with *chloroform* may be laid against the hypochondrium, and, after the part becomes accustomed to it, may be allowed to remain there, though it may be necessary to remove it every few seconds, at first, on account of the severe burning sensation it may cause. The paroxysms are very much alleviated by this local application. Large doses of *dioscorea* and *gelsemium* should be administered every hour. ℞ Specific dioscorea, gtt. xv, specific gelsemium, gtt. x. M. and administer in a swallow of water at a dose. During the paroxysms, the inhalation of chloroform may be practiced, or morphia sulph. may be injected hypodermically, though this drug hardly suffices to allay the pain, in safe doses. *Chelidonium* and *chionanthus* have been recommended as remedies for relief, though large doses of dioscorea and gelsemium will be preferable.

After the gall-stone habit has been detected, preparations of *lithium* should be regularly administered through a period of several months, to arrest their formation. Dr. Waterhouse recommends the benzoate of lithium, used by dissolving ten grains of the drug in an ounce of water and administering a teaspoonful of the mixture every three or four hours. Others recommend from three to five grains of the carbonate of lithium, stirred in a glass of water, and taken at a dose, three or four times a day. *Olive oil*, taken in tablespoonful or wine-glassful doses three or four times daily, is an old and reliable remedy for the habit. The galvanic, salt-water *electrode*, used in the lower bowel with the negative pole, the positive being applied over the right hypochondrium, is an excellent measure to promote fluidity of the bile and encourage normal biliary function. In the treatment of chronic obstruction from biliary calculi, the conditions of each case must decide the course to pursue. In serious affections of the gall-bladder, *cholecystotomy* is the proper measure.

Miscellaneous Affections of the Biliary Passages.

Cancer of the gall-bladder may occur as an independent affection, either primarily or secondarily, though there are many difficulties in the way of diagnosing it from cancer of the liver, and there is little clinical need of this. It usually arises from the irritation of impacted biliary calculi.

Stenosis of the biliary ducts may exist, either congenitally or acquired from the irritation of impacted gall-stones, though the condition is very rare in either case. When the occlusion is acquired, it usually exists low down in the common duct. Foreign bodies may obstruct the biliary passages, intestinal worms, fluke-worms, echinococci, the seeds of various fruits and other accidental substances, being most common factors.

Obstruction from pressure from without is more common than obstruction from internal causes. Carcinomatous growths from neighboring viscera are the usual causes of such obstruction. The pylorus, head of the pancreas, neighboring lymphatic glands, stomach and other abdominal organs, may be the seat of malignant growths, which exercise pressure upon the common bile-duct, to occlude it. The symptoms are those of deeply marked icterus, with or without hepatic intermittent fever; and hepatic colic, more or less severe, alternates with painless periods and gastric disturbances, with gradual progression toward a fatal termination, unless the obstruction is removable. The diagnosis is obscure, and is usually determined by an autopsy.

XI. DISEASES OF THE PANCREAS.

Hemorrhage.

Etiology.—The causes of non-inflammatory hemorrhage of the pancreas are somewhat obscure. Traumatism undoubtedly figures as a causal factor at times, and self-digestion may occasionally be responsible for it. Hemophilia and purpura may also be reckoned among occasional causes.

Pathology.—Diffused blood may invade the parenchyma of the organ, and the cellular tissue. Sometimes the hemorrhage is confined to a portion of the gland, while at other times the entire structure of the pancreas may be invaded.

Symptoms.—The principal symptom is that of marked and rapid prostration, amounting to collapse. Pain in the epigastric region may be present, and sometimes vomiting, with subnormal temperature. If death does not soon follow, inflammation sets in

about the hemorrhagic areas. Death is likely to occur within from half an hour to a few hours. Should inflammation arise the case may be somewhat prolonged, acute hemorrhagic pancreatitis being then developed.

Treatment.—Little can be expected from treatment. Stimulants, such as hypodermic injections of strychnia (gr. 1-50th every half hour until two or three doses have been administered), and brandy per mouth may be tried, to bring about reaction from the collapse. Should reaction follow and inflammation arise, the treatment will be that for hemorrhagic pancreatitis.

Acute Pancreatitis.

Synonym.—Acute Hemorrhagic Pancreatitis.

Etiology.—This may arise from traumatism, alcoholismus, gastro-duodenitis, or as a result of non-inflammatory hemorrhage of the pancreas. It sometimes follows typhoid fever, pyæmia, septicæmia, acute tuberculosis, and parotitis (metastasis). Mercury may sometimes be responsible for it, especially when its use has been prolonged.

Pathology.—The organ is hyperæmic, firm in consistency, enlarged, and its substance is infiltrated with scattered areas of small hemorrhages. Sometimes the hemorrhage is more extensive, and infiltrates the omentum and contiguous parts. If suppuration has occurred, small abscesses are found. In febrile diseases, parenchymatous changes may take place through the entire organ. Surrounding parts may be involved, abscesses arising in the surrounding connective tissue and lymphatic glands, and sometimes the pancreas may be surrounded by pus, which may finally burrow into the stomach, duodenum, peritoneal cavity or through the abdominal wall. Tension of the nerves of the cœliac plexus may give rise to intense pain. Fat necrosis seems a peculiarly common pathological condition in pancreatic disease and it is sometimes found here, the areas of necrosis varying in size from that of a pin-head to that of a hen's egg, scattered through the pancreas, omentum and other abdominal organs. *Gangrene* sometimes occurs, cases having been reported where the pancreas was entirely sequestrated, and discharged as a slough from the bowels.

Symptoms.—Pain of colicky nature, over the region of the pancreas, with prostration, restlessness and anxiety, are the leading symptoms. The pain is intense, deep-seated, and radiates to the back, shoulders and diaphragm. There is difficult and sighing respiration, with prostration, nausea and vomiting, distention of the epigastrium, clammy skin and cold sweat on the forehead. Con-

stipation is the rule, though diarrhœa may occur as a metastasis of the inflammatory action to the intestine.

Diagnosis.—The diagnosis is difficult. Acute perforative peritonitis or intestinal obstruction is more liable to be suspected. The sudden onset and intense pain—seated deeply in the pancreatic region, due, probably, to tension of the nerves of the cœliac plexus—would suggest the condition, especially if rapid prostration with vomiting and constipation supervened, though these symptoms might also be present in obstruction of the intestinal canal.

Prognosis.—Fatal results usually follow in from one to four days, though recovery occasionally results.

Treatment.—The therapeutics of acute disease of the pancreas are not in an entirely satisfactory condition. *Iodine*, in minute doses (3x or 6x), has been used with some satisfaction, and iris versicolor specifically improves the recuperative power of the part. Both remedies may be thought of and tried in cases which offer any hope of even a few hours' lease of life. Rest in the recumbent posture, and a mild and unstimulating liquid diet, must be enjoined, with such supporting measures as the extreme prostration demands. *Strychnia*, hypodermically, will be the ideal stimulant, care being exercised to employ it within efficient bounds.

When hemorrhage seems to be profuse, as indicated by symptoms of excessive prostration, *erigeron*, *rhus aromatica* or *lycopus* may be thought of. When gangrene is suspected the use of *echinacea* might be advisable.

Chronic Pancreatitis.

Etiology.—This disease is frequently an accompaniment of diabetes; whether as a coincidence, result or cause is not yet clearly established. Calculi may originate chronic inflammation and induration, as also may pressure from tumors. Chronic inflammation may invade the organ from other parts, as in ulceration of the duodenum, stomach and other neighboring viscera. Syphilitic infection sometimes gives rise to chronic inflammation of the pancreas.

Pathology.—There is increase of the interstitial connective tissue, as in cirrhosis. Increase of the connective tissue is attended by atrophy of the glandular structure, the organ becoming contracted, and firmer in consistency than normal. Closure of the duct, calculi and cystic formations in the substance of the organ may be results of constriction. Interstitial hemorrhages are likely to occur and, in the suppurative form, pus may infiltrate the organ, or one or two small abscesses may be found. Adhesions frequently bind the organ to adjacent parts.

Symptoms.—Arrest of normal function may interfere with the digestion of fats, and indigestion attended by fatty stools will naturally suggest the condition. The presence of a transverse tumor in the epigastrium, deeply seated, will add to the symptoms. Melituria, neuralgia, emaciation, etc., followed (as pressure interferes with the portal circulation) by ascites, will complete the picture.

Treatment.—When fatty stools appear, the free use of *olive oil*, with a spare diet free from fats, in which pickled olives may be allowed *ad lib.*, may prove not only temporarily beneficial, but permanently curative. Two or three ounces of olive oil should be administered at a dose, three or four times daily, until the fatty stools are replaced by healthy evacuations. Meantime, minute doses of *iris versicolor* and *iodine* may be alternated, each being taken three or four times in twenty-four hours.

If the inflammatory condition be recognized early, much benefit may follow the use of *potassium chloride* 3x, through its influence in controlling plastic exudation.

Fatty Degeneration.

Fatty infiltration and fatty degeneration both occur in the pancreas, under varying circumstances. In *fatty infiltration* the connective tissue becomes involved by the deposition of fat, and the pressure causes gradual disappearance of the gland-cells of the organ, complete disappearance of secreting structure resulting ,until the whole gland becomes a mass of fat, with the duct constituting a central canal. In *fatty degeneration* the gland-cells are primarily involved, the destruction not including the cupsule, septa and blood-vessels, which remain to constitute a soft, wasted, flaccid body, resembling the pancreas in shape, but lacking its secreting power. The causes of these conditions are similar, being alcoholism, general obesity and, in degeneration, heart disease and obstruction to the escape of the pancreatic secretion.

The symptoms are obscure, the principal ones being those which arise from gradual loss of function.

Waxy Degeneration.

Waxy degeneration of the pancreas is a very rare disease, and one of the rarest of diseases of the pancreas. It arises from the usual causes of amyloid degeneration, such as chronic ulceration of bone, prolonged suppuration, syphilis, etc. The diagnosis is obscure, and treatment is of little avail, a fatal termination within a few months being inevitable.

Cancer of the Pancreas.

Etiology.—Little is known except that the disease occurs most frequently in men after the fortieth year of age. It occurs both as a primary and secondary disease, though it is not frequent in either form.

Pathology.—Scirrhus is the common variety, and there is a tendency to involvement of adjacent organs. The head is most frequently affected first, and pressure upon the bile-duct may then occur, resulting in jaundice. The large blood-vessels in the vicinity may be obstructed, crowding upon the portal vein, giving rise to accumulation of ascitic fluid. The canal of Wirsung may be obstructed and cysts form, from tension of retained secretion. Ulceration into neighboring structures may occur, as breaking down proceeds.

Symptoms.—The symptoms are varied and often obscure, the variation depending upon the complications which arise from affection of neighboring organs. Where the head is largely involved, in thin persons, a deep tumor may be recognized in the pyloric region. Dyspeptic symptoms, intense neuralgic pains of paroxysmal character in the pyloric belt, and possibly, though not necessarily, fatty stools, are among the prominent symptoms. Rapid emaciation and loss of strength, with speedy development of cancer cachexia, soon proclaim the malignant character of the disease, which only requires to be located. The presence of free hydrochloric acid and absence of coffee-ground material in the vomit will remove suspicion of gastric cancer, and the presence of stearrhœa will confirm suspicion of disease of the pancreas. Jaundice with these distinguishing features may render diagnosis between cancer of the pancreas and liver difficult. Marked pulsation of the aorta, communicated to the epigastrium, would be a diagnostic aid in this case, suggesting an impulse communicated by the hardened pancreas.

Treatment.—Little can be done to stay the course of the disease, and death usually occurs within a year. The intensity of the suffering may be modified by the regular administration of ten-drop doses of specific *echinacea*, repeated three or four times daily. To each dose one or two drops of specific *iris* may sometimes be added with benefit. A spare and bland diet, consisting of pancreatinized meat and milk should be employed, and the patient should remain quiet in bed.

Calculi and Cysts.

Concretions in the pancreatic ducts occasionally obstruct their lumen and cause great dilatation and distention behind the point of obstruction, with atrophy of the gland structure. Pancreatic con-

cretions are usually formed chiefly of carbonate of lime, are round or oval, with rough or spinous surfaces, and present a white or opaque white color. When numerous they cause serious dilatation of the pancreatic ducts, cystic formations arising in consequence. They seldom give rise to abscess. Obscure colicky pains may attend their presence, though the diagnosis is difficult. Fatty stools and glycosuria may be present, though not necessary symptoms. Probably pancreatic calculi occasionally pass in the stools.

Pancreatic *cysts* commonly result from impaction of the ducts with calculi. Biliary calculi, lodging at the orifice of the common duct, may obstruct the duct of Wirsung, and pancreatic concretions within this duct or its branches may constitute causal factors. Other causes of obstruction of the pancreatic duct leading to similar results are cicatricial contraction af the duct of Wirsung and misplacements, by which a passage is doubled upon itself. Cicatricial contraction and misplacements may be due to injuries.

A pancreatic cyst may attain an immense size and be mistaken for an ovarian tumor, though usually it remains in the epigastric region and is perceptible upon palpation as a smooth, lobulated tumor, either occupying the median portion or one side. Aspiration of the contents of the tumor will aid in diagnosing the case, as the fluid will emulsify fat and convert starch into sugar. Disturbance of digestion, with fatty stools and glycosuria, may be present.

Sarcoma, syphilis and tuberculosis occasionally involve the pancreas.

XII. DISEASES OF THE PERITONÆUM.

Acute General Peritonitis.

Definition.—Acute inflammation of the peritonæum.

Etiology.—This disease may be (1) idiopathic (primary), or (2) symptomatic (secondary). Primary peritonitis is of rare occurrence, though it may arise from sudden chilling of the surface. To this form the term "rheumatic peritonitis" has been applied. For some inexplicable reason the peritonæum is not nearly so liable to primary inflammation as the pleura and pericardium. The usual cause of acute peritonitis is disease of some abdominal or pelvic viscus (secondary peritonitis). Traumatism, perforation of the stomach or bowel, typhlitis, metritis, ovaritis, rupture of the gall-bladder or of the cystic or common bile duct, surgical operations attended by opening in the peritoneal cavity, etc., are causes of secondary peritonitis.

Pathology.—The inflammation commonly begins in some circumscribed place and afterward becomes more or less rapidly diffused over the entire membrane. A mottled appearance is observable early, but the bright redness soon becomes general, the glistening surface of the membrane disappearing and a grayish layer of fibrillated fibrin exuding, which later becomes infiltrated with pus-cells. The bowels are inflated with gases, and, in the event of abdominal incision, they are restrained with difficulty from escaping from the opening. The subserous tissues become swollen and œdematous, and filled with migrating leucocytes and such microbic forms as the proteus vulgaris, streptococcus pyogenes, bacillus coli communis, and pneumococcus, which are also found in the exudation. Adhesion of the approximated surfaces occurs, the intestines becoming glued together, to other viscera and to the abdominal walls, the peristole becoming thus impaired. Loops, in which the bowel may be incarcerated, may form. The character and amount of the exudation varies, there sometimes being a preponderance of fibrin and sometimes a preponderance of serum. The fibrinous exudation may be so excessive and the serum so scanty as to constitute the condition sometimes termed "dry peritonitis," while in other cases there is a large amount of serous exudation, the peritoneal cavity being distended by a considerable amount of thin, watery fluid, which may be mixed with pus or blood-corpuscles. Thus, the exudation may be largely fibrinous, serous, fibrino-purulent, sero-fibrinous or sero-purulent, etc. The appearance of the fluid may vary. In some cases it may be clear and colorless; in others (sero-fibrinous) it may be yellowish; in others (purulent) thin and greenish-yellow or thick, opaque and creamy; in others (putrid) grayish-green, with putrid odor, the latter condition being the result of cancerous disease or the presence of fecal or other material due to intestinal or other visceral perforation and escape of septic fluids from the digestive cavity.

Symptoms.—The disease may develop gradually, several days being occupied in the appearance of the general and local symptoms before they are fully declared; or it may arise suddenly, this being the usual course. In the latter case a chill is apt to indicate the onset. Pain, of burning, lancinating character, is always present, localized at first, but becoming diffused with more or less rapidity all over the abdomen. Excerbations, sometimes amounting to spasms, alternate with periods of less severe suffering, though there are continual burning and shooting pains, which are aggravated by movements of the diaphragm, coughing, sneezing, vomiting, deep inspiration, and even upon motion of the body. The abdomen becomes more or less swollen, from accumulated fluids and intestinal gases, and

exceedingly tender to pressure, flexion of the lower extremities usually being resorted to to allay tension of the abdominal muscles.

Vomiting frequently occurs, and dyspnœa is an invariable symptom, this being due to the fullness of the abdomen and aggravation of pain upon forced descent of the diaphragm. The temperature, during the reaction following the chill, may rise to 104° or 105° F. for a short time, but it afterward falls to a slight elevation above normal and remains there, or at least it seldom afterward rises above 102° or 103°. Sometimes there is no appreciable elevation of temperature during the course of a case. The pulse is rapid, small and hard (wiry), often running as high as 130 or 140 per minute, and ranging from 110 to 150. The tongue is coated white at first, but becomes dry and fissured later, of deep-red color when clean and dark-brown when coated. The bowels are liable to be loose at first, though constipation soon follows. The urine is scanty and high-colored and contains a marked quantity of indican. Micturition is usually frequent, though enuresis may be present instead.

The decubitus and general appearance of the sufferer, when acute general peritonitis is established, is characteristic. The patient lies upon the back with the knees drawn up, with the abdomen greatly swollen. The skin over the face is shriveled and leaden in hue, the eyes are sunken, the nose is sharp, and there is a worn and anxious expression about the countenance. The ears are cold and drawn, and their lobes are turned out. Tympanites is marked, the intestines being distended with gases and crowded under the diaphragm, displacing the heart, liver and lungs upward, so that the apex beat may be heard in the fourth intercostal space and the usual area of liver dullness is tympanitic. Accumulation of ascitic fluid may give rise to dullness and fluctuation in the flanks.

Diagnosis.—The previous history of a case will assist materially in arriving at a correct diagnosis. A knowledge of former appendicitis, pelvic inflammation, typhoid fever, or gastric ulcer, would be suggestive, when active symptoms afterward arose. Peritonitis may be mistaken for intestinal obstruction, colic, abdominal neuralgia, enteritis, rheumatism of the abdominal muscles or organs, renal or biliary colic, suppurative cellulitis of the abdominal muscles, or the imaginary peritonitis of hysterical persons. In intestinal obstruction there is subnormal temperature, vomiting of fecal material and localized pain continually, while in peritonitis the pain soon becomes diffused. In abdominal neuralgia there is no tympany, no rise in temperature and no tenderness upon pressure, except at the root of the spinal nerve effected, while the sensation as of a cord drawn tightly about the abdomen prevails. If tympanites attends enteritis it comes on slowly, while in peritonitis it develops

rapidly; vomiting is a common and frequent symptom in enteritis, while in peritonitis it is rare and the vomit consists of spinach-green material. In abdominal rheumatism there is no disturbance of the temperature, the pain is most severe at the point of insertion of the muscles, there is no tympanites, unless the intestinal muscularis is involved and then it is not extreme, no vomiting, and no tendency to collapse. In biliary and renal colic the pain is located near the part involved, is peculiarly paroxysmal, unattended by fever, and there is absence of tympanites and tenderness. In biliary colic symptoms of jaundice appear after twenty-four hours, and in renal colic the pain radiates along the ureter to the testicle, which is retracted. In suppuration of the abdominal walls there is not such intense pain nor such marked constitutional symptoms.

Prognosis.—Acute diffuse peritonitis is usually rapidly fatal, death occurring in four or five days in many cases, and almost invariably within ten. Intense forms result in death within thirty-six hours. Feeble action of the heart, with irregular action, shallow respiration, livid pallor of the countenance, and coldness of the extremities, with high rectal temperature, indicate impending dissolution.

Treatment.—The plastic exudation is to be controlled, if possible, and for this purpose *potassium chloride* 3x should be administered in small and frequently-repeated doses. Ten grains may be added to half a glass of water, a teaspoonful being administered from this every hour. In this place the potassium chloride proves a sedative, lowering the temperature and controlling excessive action of the pulse. A pack of towels wrung out of tepid water should be applied over the abdomen and changed every hour or two. This treatment promises the best results of any known to the writer.

Some old school authors advocate the use of opium in full doses, repeated sufficiently often to maintain complete control of peristalsis, and depend upon the use of this remedy alone, in connection with packs. Where septic accumulation in the peritoneal cavity is evidently present, abdominal section and cleansing, with subsequent drainage, is practiced by many, though results are not usually very flattering. In all cases of acute diffuse peritonitis, a guarded prognosis must be made.

In puerperal and pelvic peritonitis especial attention is to be paid to the condition of the uterus and its appendages, proper douching of the uterine cavity and vagina, being considered indispensable.

Care must be exercised about the diet. If there be vomiting all attempts to administer food or drink by the mouth must be avoided, and nutrient enemata employed. Where vomiting is not present

very small quantities of predigested milk may be swallowed frequently, only a little being allowed at a time, for fear of exciting peristalsis and the accumulation of gases. If water be allowed but little should be taken at a time.

Peritonitis in Infants.

Infants are occasionally subject to peritonitis, congenital syphilis being the most frequent cause. In this instance the disease may so develop during the prenatal state as to result in constriction of the bowel from fibrinous adhesions. A cause of peritonitis in the new born is irritation of the umbilical cord, that condition extending to the abdomen and giving rise to septic peritoneal inflammation.

Peritonitis in older children is liable to result from injuries received at boisterous play, or from kicks, blows or bruises about the abdomen. It has several times been known to occur as an epidemic among children in schools, and has been attributed to the effects of sewer-gas poisoning.

The *symptoms* of septic peritonitis in the new-born are those which usually arise in other forms of peritonitis, complicated with symptoms of malignant jaundice. There is marked distention of the abdomen, shallow breathing, evident pain upon pressure and motion and marked jaundice, with, perhaps, convulsions or coma.

Little can be expected from *treatment.* Echinacea or echafolta, in minute doses, may neutralize some of the septic conditions, though the accompanying phlebitis will usually prove intractable to treatment.

Localized Peritonitis.

Pelvic Peritonitis.—This usually arises from inflammation of the uterus and Fallopian tubes, due to puerperal septicæmia, gonorrhœal infection, or tuberculosis. Sometimes, when a former inflammation has existed in the pelvis, sitting on cold surfaces or sudden chilling may give rise to the inflammatory condition leading to it. In other cases the incautious use of instruments during curettage of the uterus or other intra-uterine operations may result in pelvic peritonitis. The disease is most liable to arise in the tubes, the fimbriated extremity becoming inflamed, swollen and covered with exudate which glues the affected part to the ovary and drags surrounding tissues together into an unrecognizable mass, the broad ligament becoming infiltrated with pus, and purulent accumulation distending the Fallopian tubes, sometimes to the extent of bursting. Rupture of one of the Fallopian tubes or of an abscess of the broad ligament may cause general peritonitis. Tuberculosis of the pelvic tissues may

give rise to localized peritonitis. The symptoms are localized pain, with swelling and tenderness of the parts, slight elevation of temperature, especially in the evening, chilly sensations, and hectic fever. There is more or less obstruction of the bowels, loss of appetite and general derangement of the stomach. Throbbing sensations in the pelvis soon attend. The only successful plan of treatment is the early and steady use of *potassium chloride* 3x. When this is begun early and persevered in, the affection can usually be controlled before the formation of pus, and the inflammation subsides by resolution. Add ten grains of potassium chloride 3x to half a glass of water and order a teaspoonful every hour. Follow this day after day for three or four weeks. A liquid diet should be prescribed, and an occasional mild laxative of salts or decoction of rhamnus californica bark.

Appendicular Peritonitis.—This is the most frequent form of localized peritonitis in the male. Appendicitis has already been fully considered, and the reader is referred to that article for the pathology, symptoms and treatment.

Subphrenic Peritonitis.—The lesser peritonæum may be involved in localized inflammation arising from perforations of certain parts of the stomach, colon or duodenum, inflammation of the pancreas, or pyo-thorax. The lesser peritonæum may become distended with fluids, forming a tumor which may be mistaken for cyst of the pancreas. Accumulations of pus may occur here (perihepatic abscess), and even distention of the part with air, the latter condition being due to communication with the lung, stomach or bowel. In some cases traumatic perforations are followed by a similar condition. The symptoms are characterized by severe localized pain confined to the epigastrium, which may be abrupt in its onset, particularly when due to perforation of the stomach or bowel, and this is often accompanied by vomiting of bilious or sanious material. With these symptoms develop fever, chills, emaciation, and suppuration. Perforation into the lung is announced by cough and profuse expectoration. The prognosis in such cases is doubtful, surgical interference offering the only hopeful prospect.

Chronic Peritonitis.

Etiology.—Chronic peritonitis may follow an attack of acute peritonitis, or may arise gradually from irritation of an abdominal viscus. It is believed that it sometimes arises idiopathically, though such cases are probably really due to new growths, such as cancer or tubercle. The following varieties of chronic peritonitis may be

mentioned: (1) Local adhesive, (2) diffuse adhesive, (3) proliferative and (4) hemorrhagic.

Local adhesive peritonitis arises as secondary to inflammatory disease of one of the abdominal viscera. Its favorite locality is about the liver and spleen. The symptoms are not marked, though persistent abdominal pain may attend some cases, others occasioning no inconvenience and being overlooked, unless discovered accidentally upon autopsy. Sometimes loops are formed by adhesions, into which the intestines may be incarcerated, giving rise to intestinal obstruction.

Diffuse adhesive peritonitis results from acute inflammation of the peritonæum. Here the peritonæum may be completely obliterated, the visceral and abdominal layers being welded together and the intestines matted, the adhesions usually involving the liver and spleen.

Proliferative peritonitis is characterized by remarkable thickening of the peritonæum, without adhesion of its surfaces. It occurs in cancer and tuberculosis of the peritonæum, and in cirrhotic conditions of the liver or portions of the intestinal canal, often in subjects of chronic alcoholism. The peritonæum is white and opaque in appearance, generally thickened, though there are patches where the thickening is greatly exaggerated. About the liver and spleen this may be marked, a layer of gristly connective tissue half an inch or more in thickness sometimes enveloping these organs. Constriction of the inclosed viscera attends, and they become much reduced in size. Sometimes the constriction results in obstruction to the portal vein. Thickening of the intestinal walls may occur, and the abdominal viscera may be drawn up into a ball not much larger than a child's head. There may be moderate effusion and sometimes marked ascites, though in other cases the peritonæum may be divided into several sacs, each containing circumscribed fluid. Friction-sounds in these cases are usually heard in the upper portion of the abdomen. Nodular thickening has been observed in rare cases, which has been determined to be neither tubercular nor cancerous, and which has been supposed to be due to the presence of parasites. Nodules of this character may be disseminated through the liver. A Japanese investigator asserts that the nodules contain the ova of a parasite.

Chronic hemorrhagic peritonitis is characterized by the successive formation of new connective tissue upon the surface of the peritonæum containing open blood-vessels, from which exude blood-stained effusions. The hemorrhagic formations are usually circumscribed, and commonly occur in cancer and tuberculosis.

Treatment.—With our present means of treatment little can be

done for chronic peritonitis, except to keep the patient quiet and enjoin the use of a spare animal diet, with almost complete avoidance of vegetables and starchy food. *Potassium chloride* 3x may sometimes assist in controlling plastic exudation, especially when used patiently and persistently for a long time. An important step is the prevention of ascitic accumulations in the intestines, and careful attention to diet will accomplish the most of this, vegetables, sugars and starches being objectionable. Broiled tender chops, steaks, white meat of fish or chicken, in small quantities and carefully and slowly masticated, are best adapted. When the stomach digests them eggs, milk and cream may be taken. To alleviate the formation of gases half a drachm of *listerine* or five grains of *eudoxine* may be administered half an hour or so after eating.

New Growths in the Peritonæum.

The peritonæum may be the seat of tubercular and cancerous growths, as well as of nodules resulting from the presence of echinococci.

Tubercular peritonitis may be primary and local. It may attend tuberculosis of the lungs or follow an attack of acute miliary tuberculosis. It is common in children, more frequent in males than in females, and is most apt to be found between the ages of twenty and forty, though it may occur in advanced life. Extensive thickening and adhesions occur, the omentum being puckered and bunched and drawn across the upper portion of the abdomen. Sacs are formed, in which accumulations of sero-purulent or purulent material are found, the amount varying, though the entire abdomen may be distended, as in ascites. Localized abdominal tumors may represent smaller collections. General wasting of flesh, digestive disorders, and more or less abdominal pain are the leading symptoms. The temperature is remarkably prone to be subnormal, a morning range of 95.5° F. often being found. *Treatment* can accomplish little here, palliative measures only being applicable. Opiates, dietary regulations and, in some cases, surgical relief of distended accumulations are the principal means to be relied upon.

Cancerous growths in the peritonæum may be primary or secondary, the latter condition being the rule and simplifying the diagnosis. When occurring secondarily the stomach or ovary is usually the starting point. Cancerous nodules are distributed over the peritoneal surface, and the omentum becomes puckered and drawn up as in tuberculous peritonitis, forming a transverse tumor across the upper portion of the abdomen. The disease is disseminated by contact of opposing surfaces (transplanted) or through lymph currents

which carry the cancer cells to different parts. The *diagnosis* is not difficult when the disease is secondary to localized cancer, but when primary there is so much resemblance to tuberculous peritonitis in many respects that there may be confusion. Cancer, however, presents more marked nodulation, this being apparent on palpation when ascitic fluids have been evacuated. There is greater pain in cancer usually, and the cancerous cachexia is more or less apparent. *Treatment* is not highly satisfactory. *Echinacea* may be tried for the pain, paracentesis will relieve oppressive distention of the abdomen, and rest and proper diet may assist in prolonging life.

Echinococci give rise to nodular growths on the peritonæum, these occurring in connection with hydatids of the liver. There is not much danger of confounding this affection with cancer or tubercle, as the general health is not much involved, the principal trouble arising from pressure of the morbid growth. The enlarged liver will originate the leading symptoms, which will be mechanical.

Ascites.

Synonyms.—Abdominal Dropsy; Hydroperitonæum.

Definition.—An accumulation of serum in the abdominal cavity.

Etiology.—Ascites may occur during the late period of general dropsy, though it usually arises from portal obstruction, the *vis a tergo* from the abdominal arteries then forcing the serum from the capillaries into the peritoneal cavity. All forms of peritoneal inflammation are attended by more or less effusion into the peritoneal cavity due to capillary changes, and sometimes the amount may be sufficient to distend the peritoneal sac to its utmost limits. Diseases of the heart or lungs which contribute to obstruction in the venæ cavæ may be attended by ascites, though general dropsy is more liable to attend such condition. The common causes of ascites are those which give rise to portal obstruction, such as cirrhosis of the liver, hepatic cancer, biliary obstruction, pressure from tumors or cicatricial bands and thrombus of the portal vein. Asthenic conditions may be attended by ascites when there is no obstruction to the circulation, as in hydræmic states of the blood due to anæmia, chlorosis, malarial cachexia, purpura, chronic arsenical poisoning, chronic Bright's disease, and senility or great exhaustion, the condition then being considered asthenic or *cachectic ascites*. Sudden arrest of secretion from chilling of the surface, especially during menstruation, or the sudden suppression of cutaneous affections, may be followed by it. Tuberculous or cancerous disease of the peritonæum is apt, after degenerative changes have become well established, to be attended by dropsy.

Pathology.—The endothelia of the peritonæum are swollen, and manifest more or less fatty degeneration. They appear turbid, and the subserous tissue is increased in bulk, the entire membrane appearing soggy and inelastic. The fluid in the abdominal cavity varies from a few ounces to five or six gallons. It may be viscid or watery in consistence, and is usually of a yellowish straw color with an opalescent greenish tint, though if there be an admixture of blood it may be dark, while in disease of the lymphatics it may be milky. Sometimes it is as clear and as limpid as water. Chylous fluid may depend upon perforation of the thoracic duct from cancerous disease, or upon filariæ. The specific gravity varies from high to low, though it is usually as low as 1.010 or 1.015. The blood of ascitic patients is usually hydræmic and poor in albumen.

Symptoms.—Gradual increase in the size of the abdomen is the characteristic symptom of ascites. With this will be associated the symptoms which attend the particular etiological factor of each individual case. Portal obstruction, various forms of peritonitis, cachexiæ and other provoking conditions manifest themselves in conjunction. Where there is biliary obstruction there may be marked symptoms of jaundice. In other cases the patient may present a pallid appearance, or the waxy color of cancer may be prominent.

Physical signs are important as diagnostic symptoms. *Palpation* imparts a peculiar wave to the fingers, which, without doubt, attests the presence of fluid in the cavity. The fingers of one hand should be placed upon one side of the abdomen while a sharp tap is given upon the opposite side with the other hand. A distinct wave passes across and imparts its shock, whenever there is fluid in the abdomen, to the stationary fingers. *Percussion* elicits information of further value; change of position alters the relative location of the fluids, which impart dullness, and the intestines, which give resonance. In the upright position the fluid gravitates to the lower part of the abdomen, and the intestines rise toward the diaphragm, dullness being found upon percussion of the lower part of the abdomen and resonance when percussion is made higher up over the intestines. When the patient is in the dorsal position the dullness will be in the flanks and the tympanitic sounds over the middle-line of the abdomen. When the patient is turned upon the side the dullness will be over the lower flank and the tympanitic sound over the upper one. Change of position will thus be followed by change of location of dullness and resonance, corresponding to the shifting of the fluid and intestines due to gravity. In case there is a very small amount of fluid the knee-chest position may be necessary to detect it.

Ascites should be differentiated from a large ovarian tumor cen-

trally placed, which remains fixed centrally and pushes the intestines into the flanks. Here the points of dullness are reversed when the patient is in the dorsal position from those of ascites. A distended bladder may be mistaken for abdominal dropsy, and the awkward act of plunging a trocar into the viscus has been committed by surgeons in the past, though this may seem an incredible error. The condition of the bladder should always be determined before the operation of paracentisis. Pancreatic and hydatid cysts have been confused with ascites, though such errors should not occur with careful attention to diagnostic points.

Treatment.—The treatment of ascites should be adapted to individual cases. The causal or provoking factor should receive first attention. Portal obstruction, if amenable to treatment, should be removed, and, in malarial cachexia, spleen remedies should be administered to assist normal portal circulation. Hydræmic conditions may require *calcium phos.* 3x, *calcarea carb.* 3x, preparations of iron, change of climate or other provision. The diet should be generous, unless digestion is seriously impaired, and as small an amount of fluid as possible should be taken. Heroic measures for the removal of abdominal fluids should be avoided. Hot or steam baths are not adapted to this condition. Active cathartics seldom accomplish permanent good, and prove debilitating from the start. Sometimes, when there is not serious organic disease present, cathartic doses of *elaterium*, employed for a few hours at intervals, remove the fluid, and judicious after-management prevents its return. The use of diuretics and diaphoretics is favored by many, but such measures seldom accomplish much. *Apocynum cannabinum*, in five- or ten-drop doses of the specific medicine, four or five times daily, may prove successful. In other cases benefit might follow the use of *convallaria majalis*, in five-drop doses repeated every three or four hours during the day. Sometimes, when the accumulation is due to arrested cutaneous exudation, a change of climate will serve the purpose. In the case of an old sea captain with enormous ascitic accumulation, a prolonged residence in the interior of California resulted in a permanent cure. Of course, in this case there was no organic disease or portal obstruction.

Most cases finally become so distressing from distention as to need paracentisis, and this may require repetition a number of times, whatever the ultimate result. Important considerations in such cases are precautions as to asepsis of instruments employed, and care not to injure any of the abdominal viscera.

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